

**PERCUTANEOUS TRANSHEPATIC ACCESS
TO PORTAL VENOUS AND BILE DUCT
SYSTEMS FOR DIAGNOSTIC
AND THERAPEUTIC PROCEDURES**

Jürgen Hoevels

Lund 1979

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PERCUTANEOUS TRANSHEPATIC ACCESS TO PORTAL VENOUS AND BILE DUCT SYSTEMS FOR DIAGNOSTIC AND THERAPEUTIC PROCEDURES

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The topographic relationship of the portal vein to the extrahepatic bile ducts was investigated by simultaneous portography and cholangiography at autopsy. In the porta hepatis the main hepatic duct junction was found to be in direct contact with the portal vein or its branches, whereas minor variations in the interspace between the bile ducts and the portal vein were demonstrated in the hepatoduodenal ligament and the duodeno-pancreatic region. The findings were discussed as to their significance in cases of carcinoma of the extrahepatic bile ducts.

The diagnostic value of PTC, PTP and angiography in patients with carcinoma of the extrahepatic bile ducts was studied. PTC was found to be an indispensable method in the demonstration of tumor involvement of the biliary ducts in cholangiocarcinoma. In the majority of cases both angiography and PTP were found to be insufficient in indicating the extent of tumor growth outside the biliary ducts and of tumor resectability.

The efficacy of the percutaneous transhepatic insertion of a permanent bile duct endoprosthesis was evaluated in patients with obstructive lesions of the extrahepatic bile ducts. The technique, complications and clinical application were discussed.

The diagnostic value of PTP was evaluated in patients with malignant lesions of the liver. The findings at PTP were correlated with those of infusion hepatic angiography.

Referat skrivet av PTP was found to be far inferior in diagnostic effectiveness to author infusion hepatic angiography in space-occupying lesions of the liver.

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J Ü R G E N H O E V E L S

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This thesis is based on the following papers:

- I. Topographic relation of portal vein to extrahepatic bile ducts.
A combined portographic - cholangiographic study in 25 cadavers.
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- II. Percutaneous transhepatic portography in bile duct carcinoma.
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- III. Percutaneous transhepatic insertion of a permanent endoprosthesis in obstructive lesions of the extrahepatic bile ducts.
J. Hoevels and I. Ihse
Submitted for publication in Gastrointest Radiol (1979)
- IV. A comparative diagnostic study of malignant lesions of the liver by infusion angiography and percutaneous transhepatic portography.
J. Hoevels
Accepted for publication in Fortschr Röntgenstr (1979).

Reference to these papers will be made by their Roman numerals, I - IV.

ERRATA

- Paper II, page 1, English abstract, line 9 reads "... in 4 patients".
Should read "... in 5 patients".
- Paper II, page 1, German abstract, line 18 reads "...bei 4 Patienten".
Should read "... bei 5 Patienten".
- Paper II, page 11, second column, line 3 reads "...from the arterial phase alone in one patient and from the arterial and venous phases in 2". Should read "...from both the arterial and venous phases in these 3 cases".
- Paper II, page 11, second column, line 7 reads "... in 4 patients."
Should read: "... in 5 patients".

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INTRODUCTION

Direct access to the portal venous system by percutaneous transhepatic puncture and catheterization (PTP) is by now a widely accepted radiographic method. It is apparent from the literature that interest in this technique is focused mainly on its value in diagnostic and therapeutic investigations in patients with portal venous hypertension and in the diagnostic aspects of selective blood sampling for hormone assay studies in patients with endocrine gastro-intestinal tumors. Less attention has been paid to the diagnostic value of PTP in patients with malignant tumors in the hepato-biliary-pancreatic region. It was therefore considered advantageous to examine the yield of PTP as a complement to standard angiographic examinations in patients with carcinoma of the extrahepatic bile duct and malignant liver tumors.

The percutaneous transhepatic approach to the bile duct system developed an interesting therapeutic aspect when a technique for non-surgical introduction of a bile duct endoprosthesis became available. This method is highly invasive and its usefulness must be balanced against the possible liver damage to which the patient is exposed. Analysis of even a limited material was therefore regarded as a necessary initial step in the further elaboration of this method.

The purposes of the present investigations have been:

1. To study the topographic relation of the portal vein to the extra-hepatic bile ducts in the porta hepatis, hepatoduodenal ligament, and periampullary region (I),
2. To determine the diagnostic value of percutaneous transhepatic portography (PTP) in indicating tumor extirpability in patients with bile duct carcinoma (II),
3. To evaluate the efficacy of the percutaneous transhepatic insertion of a permanent bile duct endoprosthesis, its technical aspects and clinical application (III),
4. To assess the diagnostic effectiveness of PTP compared with infusion angiography in patients with primary and metastatic tumors of the liver (IV).

HISTORICAL BACKGROUND

PTP

Percutaneous transhepatic access to the portal venous system was introduced by BIERMAN et coll in 1952. By insertion of a needle from the epigastrium, an intrahepatic branch of the portal vein was punctured in order to obtain blood samples, make pressure readings, and inject contrast medium for radiographic examinations. In a subsequent investigation by STEINBACH et coll (1953) of the angiographic aspects of direct portal vein puncture, some disadvantages of the procedure were apparent:

1. Only the intrahepatic portal venous system could be demonstrated,
2. Extravasation of contrast medium resulted from the technique applied,
3. Insertion of a needle through the liver parenchyma was necessary.

The approach to the portal venous system without laparotomy was regarded as the main advantage of the procedure. Most of the previous methods of direct venography had required an internal approach which necessitated laparotomy. Contrast media had thus been injected into a branch of the coronary vein (BLAKEMORE and LORD 1945), into a small vein of the cecum or mesoappendix (DeSOUSA PEREIRA et coll 1949), into the portal vein with a catheter inserted via the right gastroepiploic vein (MOORE and BRIDENBAUGH 1950, 1951) or through a catheter placed in the superior mesenteric vein (CHILD et coll 1951).

NUNEZ et coll in 1954 reported percutaneous transhepatic puncture of intrahepatic portal vein branches from the right mid-axillary line for direct measurement of the portal venous pressure and radiographic examination. BIERMAN et coll (1955) described an improved technique of transhepatic portal vein puncture and catheterization of an intrahepatic portal vein branch. A small ureteral catheter or hardened polyethylene tubing was threaded through the needle into a portal vein branch as the needle was withdrawn, leaving the tubing in place in the vessel. The technique was designed to obtain blood samples over prolonged periods. The authors concluded that catheterization was inadequate for portography because of the small caliber of the catheter.

The direct approach to portal venous circulation without laparotomy, for determination of the patency of splenorenal shunts, was reported by SILVA et coll in 1961. A polyethylene catheter was advanced through the puncture needle into the main stem of the portal vein or into the splenic vein for contrast medium injection. Although the narrow tubing did not permit rapid administration of the contrast medium, the patency of the shunts could be demonstrated in four cases with the catheter inserted into the splenic vein. ARNER et coll in 1963 and 1965 described the method of percutaneous transhepatic puncture and catheterization of intrahepatic portal vein branches from a point below the right costal margin. The procedure was used for the recording of portal vein pressure. No radiographic examination was made of the portal venous system.

Refinement of the technique of percutaneous transhepatic portography (PTP) towards selectivity was described by WIECHEL in 1971 (a). He used the lateral approach from the right mid-axillary line and applied the catheter-guidewire technique, which made possible selective catheterization of the portal venous system and its tributaries. Experience with this method in larger series of patients was reported by VIAMONTE et coll (1975, 1977 b), RUSSELL et coll (1976, WIDRICH et coll (1976), HOEVELS (1978), HOEVELS et coll (1978 a), BURCHARTH and RASMUSSEN (1974), and BURCHARTH (1979). LUNDERQUIST and VANG (1974 a, b) in-

troduced a treatment of acute variceal hemorrhage by percutaneous transhepatic catheterization and occlusion of porto-systemic collaterals draining esophageal varices. Since then numerous investigations have been published describing the use of the selective percutaneous transhepatic access to the portal and esophageal veins for diagnostic and therapeutic purposes (DOYON et coll 1975, GÜNTHER et coll 1976, 1977, SCOTT et coll 1976, LUNDERQUIST et coll 1977, 1978, PEREIRAS et coll 1977, VIAMONTE et coll 1977 a, WIDRICH et coll 1978 a, b, KELLER et coll 1978, DEIMER et coll 1978, ROCHE et coll 1979, BENGMARK et coll 1979, HOEVELS et coll 1979 a). The flow pattern of the portal venous circulation and the development of portosystemic collaterals in patients with portovenous hypertension were evaluated by percutaneous transhepatic catheterization of the portal vein and its main tributaries by VIAMONTE et coll (1977 b), KINKHABWALA et coll (1977), OKUDA et coll (1977), SIMERT et coll (1978), OHKUBO et coll (1978), NUNEZ et coll (1978), REICHARDT et coll (1979 a), BURCHARTH et coll (1979), HOEVELS et coll (1979 b, c).

The selective access to the splanchnic veins via the percutaneous transhepatic route was used for evaluation of the normal anatomy of the pancreatic veins by GÖTHLIN et coll (1974), LUNDERQUIST and TYLÉN (1975), REICHARDT et coll (1978), of the mesenteric veins by REICHARDT et coll (1979 b), and of the venous changes due to malignant tumors within or adjacent to these structures by LUNDERQUIST and TYLÉN (1975), REICHARDT et coll (1978), REICHARDT et coll (1979 b).

PTC

BURCKHARDT and MÜLLER in 1921 reported studies of percutaneous transhepatic puncture of the gallbladder in cadavers and in a small number of patients. Injections of contrast medium resulted in visualization of both the gallbladder and the intra- and extrahepatic bile ducts in the post mortem studies, whereas only the gallbladder was demonstrated in vivo. The percutaneous transhepatic puncture of the intrahepatic bile ducts for diagnostic cholangiography was described first by HUARD et coll in 1937. When CARTER and SAYPOL (1952) and LEGER et coll (1952) published

their experiences with percutaneous transhepatic cholangiography (PTC) 15 years later, they initiated an era of extensive investigation of the techniques, indications and complications of PTC. Comprehensive contributions in this field were presented by ALVAREZ and JENSEN (1953), MANDL (1956), REMOLAR et coll (1956), KAPLAN et coll (1961), ARNER et coll (1962), GLENN et coll (1962), SHALDON et coll (1962), BOROW(1964), WIECHEL (1964), WENZ and KOLIG (1965), MUJAHED and EVANS (1966), BAYINDIR (1966), PEIPER et coll (1967), MARIONS and WIECHEL (1974).

The latest and apparently the most essential step towards technical refinement was made by the Japanese authors OHTO et coll (1973), OKUDA et coll (1974), ARIYAMA et coll (1978), who advocated PTC with fine-needle technique.

An overwhelming number of publications on the percutaneous transhepatic approach to the bile ducts dealt mainly with its diagnostic aspects. LEGER et coll, however, as early as 1952 and 1953, described a combination of diagnostic transhepatic approach to the bile duct system with preoperative external bile drainage in extrahepatic cholestasis caused by a malignant tumor. Subsequently various techniques have been described for external bile drainage by percutaneous transhepatic intubation of the bile ducts, mainly in patients with malignant tumors and extrahepatic cholestasis (GLENN et coll 1962, AHNLUND and MORALES 1963, KAUDE et coll 1969, MARIONS and WIECHEL 1964, LANG 1974, TAKADA et coll 1976, TYLÉN et coll 1977, MORI et coll 1977, HENRY et coll 1978, BURCHARTH and NIELBO 1976, HANSSON et coll 1979).

Percutaneous transhepatic intubation and bile duct decompression because of strictured bilio-digestive anastomosis have been reported by ROSENMAN et coll (1977) and MOLNAR and STOCKUM (1978).

More recently the therapeutic efficacy of bile drainage via a non-surgically introduced catheter has been further improved. By manipulation of a guide wire and a drainage catheter through the obstructing lesion, combined internal/external bile flow is made possible (MOLNAR and STOCKUM 1974, HOEVELS et coll 1978 b, c, PROBST et coll 1978, ARIYAMA et coll 1978, NAKAYAMA et coll 1978, RING et coll 1978, GÜNTHER et coll 1979).

PREVIOUS INVESTIGATIONS

I. Extensive post mortem investigations have been made to evaluate the anatomy of the portal vein and its tributaries (DOUGLAS et coll 1950, GILFILLAN 1950, ROUSSELOT et coll 1953, MICHELS 1955). The intra- and extrahepatic bile duct system has been studied by both post mortem and clinical (i.e. radiographic) methods (REX 1888, DESCOMPS and LALAUUBIE 1910, BELOU 1915, NUBOER 1931, HJORTSJÖ 1948, 1950, NORMAN 1951, ELIAS and PETTY 1952).

The topographic relationship between the portal vein and the bile ducts in the hilum of the liver has been investigated by REX (1888) and BELOU (1915). The topography of the vessels and the bile ducts in the hepatoduodenal ligament has been described in standard textbooks of anatomy (CALLANDER 1939, PERNKOPF 1943, CORNING 1944, MICHELS 1955, HAFFERL 1957, WARWICK and WILLIAMS 1973). However, radiographic studies in cadavers, providing information on the actual interspace and the normal spatial variation between the portal vein and the bile ducts along their course from the porta hepatis to the periampullary region, have not previously been made.

II. Selective examination of the celiac, hepatic, gastroduodenal, and superior mesenteric arteries, is the standard angiographic procedure for preoperative determination of tumor spread and resectability in patients with suspected carcinoma obstructing the extrahepatic ducts. BOIJSEN and REUTER (1967), BAYINDIR (1968) and GÖTHLIN et coll (1973) evaluat-

ed the diagnostic yield of combined PTC and angiography in patients with extrahepatic cholestasis caused by a malignant tumor. MILLER et coll (1966) and SATO et coll (1967) combined transhepatic cholangiography with splenoportography for diagnosis in patients with jaundice caused by tumor occlusion of the extrahepatic bile ducts. No investigation of the possible value of PTP in this field has previously been made.

III. The most advanced method for non-surgical bile duct decompression was reported by WIECHEL (1976 a), BURCHARTH (1978), BURCHARTH et coll (1979), and PEREIRAS et coll (1978). A bile duct endoprosthesis without connection with the body surface was introduced via the percutaneous transhepatic access. Experience with this technique is so far limited. The largest group of patients treated with a bile duct endoprosthesis was reported by BURCHARTH et coll in 1979.

IV. Conventional selective hepatic angiography and infusion hepatic angiography have proved to be highly reliable diagnostic methods in vascular tumors of the liver (BOIJSEN 1965, WISE and SIBER 1968, WILLIAMS and WISE 1970, KAUDE et coll 1973, WIRTANEN 1973, FREENY et coll 1976). The detection of angiographically hypo- or avascular lesions of the liver is essentially less successful. Studies have been made on the diagnostic value of splenoportography (ANACKER et coll 1957, RÖSCH 1959, ARNER and FERNSTRÖM 1965), and transumbilical portography (KESSLER 1967, BOLLAERT et coll 1970, MATEEV 1972, MATEEV and WIRBATZ 1976) in tumors of the liver. No such investigation has previously been reported on the diagnostic effectiveness of PTP in liver tumors in comparison with other angiographic methods.

PRESENT INVESTIGATIONS

Radiography of the portal vein and biliary ducts in cadavers and patients with bile duct carcinoma (I + II).

MATERIAL AND METHODS

Post mortem studies

During routine autopsy of 25 adult cadavers a branch of the superior mesenteric vein within the mesenteric root was dissected and cannulated with a 0.4-cm polyethylene catheter. The tip of the catheter was advanced into the main stem of the portal vein or into one of its main branches. The catheter was fixed with a tight ligature around the cannulated superior mesenteric vein. Exploration of the bile ducts was performed either through insertion of a 0.2-cm catheter into the papilla of Vater, secured by a ligature, or introduction of a 0.4-cm tube into the gallbladder. Opacification of the intra- and extrahepatic biliary system as well as the cranial part of the superior mesenteric vein, the splenic vein, and the portal vein system was achieved by injection of 30 - 50 ml and 70 - 100 ml of barium sulphate suspension (0.6 g/ml), respectively. Films in a grid cassette were placed directly under the cadaver and exposed with 80 kV/80 mAs (0.6 mm² focus), FFD 90 cm. In addition to films in anterior-posterior projection, oblique projections were taken for evaluation of the topographic relationship between the opacified bile ducts and veins. The distance between the extrahepatic segments of the bile ducts and the portal vein and their ventral topographic relationship was investigated in:

- a. the porta hepatis,
- b. the hepatoduodenal ligament, and
- c. the duodenopancreatic region.

Clinical studies

Angiography, PTC, and PTP were analyzed and compared as to their validity in indicating tumor extirpability in 14 patients with carcinoma of the extrahepatic bile ducts. In two additional patients only PTC and PTP were performed. PTC, angiography, and PTP were carried out within three weeks prior to operation.

PTP

The PTP technique was the same as that previously described (HOEVELS 1978, HOEVELS et coll 1978a). The patient was premedicated with 10 mg diazepam and 5 mg atropine intramuscularly. Guided by fluoroscopy and a survey film of the upper abdomen, the approximate location of the hilum of the liver was estimated. This was often facilitated by gas in the duodenal bulb. Interposition of the hepatic flexure of the colon between the liver and the abdominal wall was excluded by this means. The examination was performed with the patient supine. The puncture site was chosen in the right mid-axillary line corresponding to the estimated level of the porta hepatis. Local anesthesia was applied in the subcutaneous tissue, the intercostal muscles, near the intercostal nerve and the parietal peritoneum, allowing the anesthesia needle to penetrate the liver capsule and enter its parenchyma about one centimeter. Following penetration of the skin and intercostal muscles with a large-bore needle, the puncture was performed cranially to the lower rib of the chosen costal interspace, thus preventing bleeding from the intercostal vessels. A 25-cm stylet fitted with a polyethylene catheter (ID/OD 1.0/1.6 mm) was used. The puncture was performed quickly and without interruption during fluoroscopy, towards the estimated position of the hilum of the liver in a plane parallel to the table top. The puncture should not be deeper than the estimated location of the porta hepatis. After reaching this point, the stylet was immediately withdrawn, leaving the catheter in place (Fig. 1 a). During this manoeuvre the patient was asked to hold his breath or,

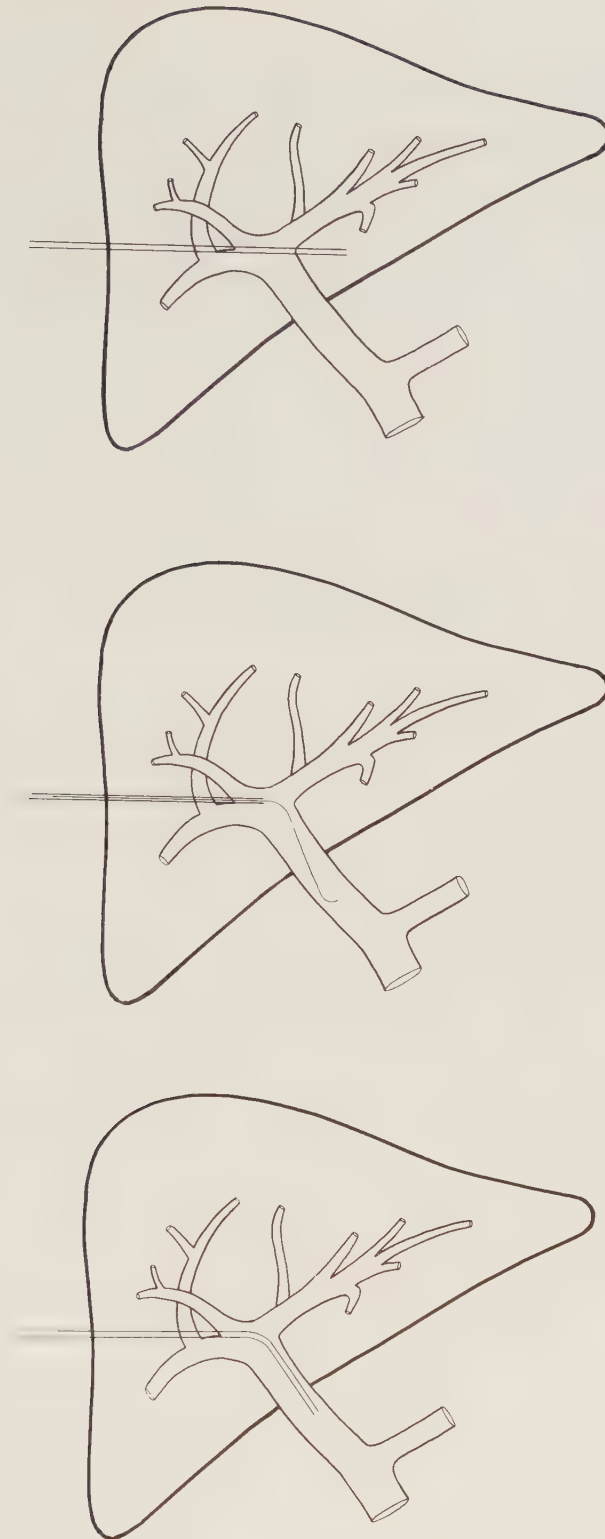


Fig. 1. a. (top) Catheter in region of porta hepatis. Stylet removed.
 b. (middle) Tip of catheter in main stem of portal vein. Guide wire with curved tip inserted and placed in proximal part of portal vein.
 c. (bottom) Catheter advanced into same position as in b. Guide wire removed.
 (With permission from Acta Radiol /Diagn/ 19(1978), 643: J.Hoevels et coll: Percutaneous transhepatic portography).

if this was not possible, to breathe gently. After removal of the puncture needle the polyethylene catheter was withdrawn slowly and stepwise until blood could be aspirated. A 10-ml syringe filled with heparinized saline was connected to the catheter. Easy aspiration of blood and easy flushing with saline indicated a satisfactory position of the tip of the catheter within the blood vessel. To identify the position of the catheter and the nature of the punctured vessel, a few milliliters of 60 per cent contrast medium was injected during fluoroscopy. If the catheter was located within the main stem of the portal vein or the right portal vein branch, a guide wire with a slightly curved tip (Fig. 2 a) was advanced through the catheter until its tip reached the spleno-mesenteric junction (Fig. 1 b). If a portal vein branch was not found while the catheter was retracted from the central part of the liver, the stylet was reinserted before the catheter passed out from the liver parenchyma, and a new puncture was performed directing the stylet towards the hilum of the liver. Thus additional puncture holes of the capsule of the liver were avoided and the risk of bleeding into the abdominal cavity was reduced. The reinsertion of the stylet must be performed with great care to avoid perforation of the wall of the catheter. The stylet could be reinserted, even if the catheter was kinked between the abdominal wall and the liver, by asking the patient to hold his breath in a position straightening the course of the catheter. The number of puncture attempts should not be rigidly limited. In general two or three puncture trials were performed; in single cases up to 10 punctures were necessary. Puncture of the more ventrally located left branch of the portal vein or its sub-branches made selective catheterization of the mesenteric vein, pancreatic veins, and left and short gastric veins more difficult. Therefore, catheterization of the main left branch of the portal veins was avoided and, instead, repuncture in a more dorsal direction was attempted in order to find a right portal vein branch. Even puncture of a second- or third-order sub-branch allowed successful catheterization of the main stem of the portal vein but the catheterization procedure in such a case was more difficult. When the guide wire had entered a larger branch of the portal venous system it could usually be manipulated into the main stem of the portal vein. The catheter was then pushed over it into the same position (Fig. 1c). Although catheterization of the main stem of the portal vein was possible by passing several sub-branches, it was usually preferable to perform an-

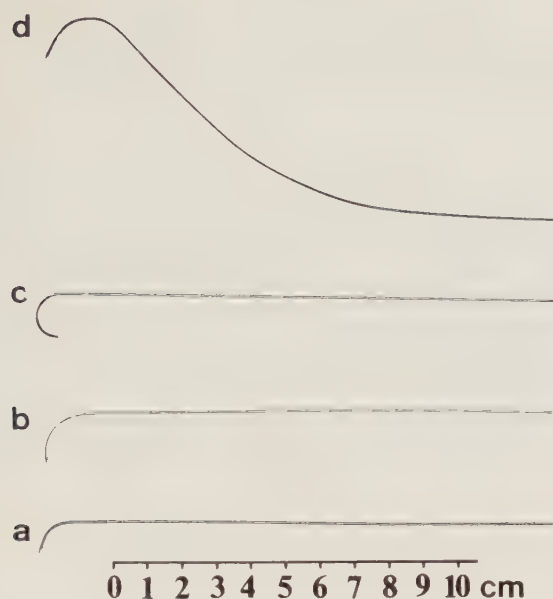


Fig. 2. Different shapes of guide wire may facilitate catheterization of portal venous system and its tributaries.

- a. 0.9 mm guide wire with slightly bent soft tip.
- b. 0.9 mm guide wire with somewhat more curved tip.
- c. 0.9 mm J-guide wire with 12 mm curve and movable core.
- d. 0.9 mm guide wire with markedly curved tip and slight bending of proximal 10-15 cm to facilitate catheterization of splenic vein when joining portal vein at acute angle.

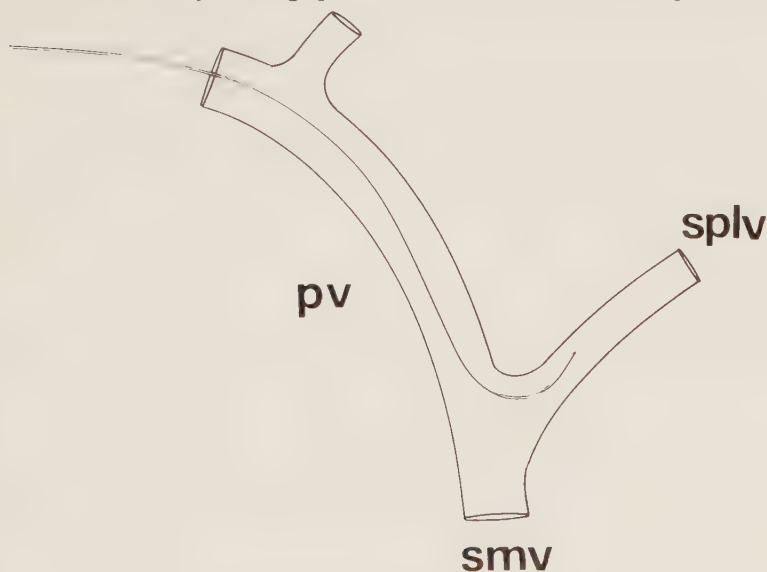


Fig. 3. Guide wire illustrated in Fig. 2 d in portal vein and splenic vein. Pv = portal vein. Splv = splenic vein. Smv = superior mesenteric vein.

(With permission from Acta Radiol /Diagn/ 19(1978), 643: J. Hoevels et coll: Percutaneous transhepatic portography).

other puncture towards the hilum in order to find the right branch or the main stem of the portal vein.

The choice of guide wire was of great importance in the performance of selective catheterization of branches of the portal vein as well as its tributaries. The best type seemed to be a straight 0.9 mm guide wire with a soft tip which could be shaped by hand. Different shapes of guide wires were chosen, depending on the anatomy and the entrance site of the catheter in the portal venous system (Figs. 2, 3).

For the examination of the intrahepatic branches of the portal vein and the parenchyma of the liver, the tip of the catheter was placed in the proximal part of the main stem of the portal vein. With the patient in the supine and right posterior oblique position, two series of films (5 films/5 s, 8 films/4 s, 3 films/6 s; 40 ml contrast medium 370 mg I/ml; flow rate 8 ml/s) with vertical beam were taken. Optimum demonstration of the left liver lobe was achieved by selective catheterization of the left branch of the portal vein. Because of the acute bending when branching off from the main stem of the portal vein, selective catheterization of the left branch must be performed cautiously. The tip of the catheter should not be directed against the wall of the vessel if a subintimal deposition of contrast medium is to be avoided (20 - 30 ml contrast medium, 370 mg I/ml; flow rate 4 - 7 ml/s).

When the examination was completed and the catheter withdrawn, a small piece of gelfoam was injected through the catheter into the parenchyma beneath the surface of the liver to avoid bleeding. This must be performed cautiously to prevent the gelfoam from passing through the puncture canal into the portal vein branch. It is best accomplished by placing the piece of gelfoam at the tip of a 2-ml syringe filled with contrast medium. The gelfoam particles were injected through the catheter and were seen during fluoroscopy approaching its tip. When the gelfoam was about 2 to 3 cm from the tip of the catheter, a guide wire was introduced and used to push the gelfoam out of the catheter into the parenchyma of the liver.

Contraindications

In portal vein occlusion PTP is contraindicated. Indirect (=arterial) portography therefore should always be performed before the transhepatic procedure to determine that the portal vein is patent. Percutaneous transhepatic portography should not be performed in patients with a platelet count below $50,000/\text{mm}^3$ because these patients represent a high risk of bleeding into the abdominal cavity during and after the examination. In emergency cases, however, when other methods to stop bleeding from esophageal varices have failed, PTP in order to obliterate veins can be performed in spite of severe coagulopathy.

PTC

After local anesthesia of the skin and intercostal muscles, a 15-cm needle (OD 0.9 mm) was inserted during fluoroscopy from the right midaxillary line parallel to the table top towards the estimated position of the hilum of the liver. During steady contrast injection into the liver parenchyma, the needle was slowly withdrawn until its tip entered a bile duct. In case of free flow of contrast medium to the duodenum, the needle was removed after radiographic demonstration of the findings. If dilatation of the bile duct was present, a sufficient amount of contrast medium was injected to demonstrate the intrahepatic bile ducts, whereafter the needle was withdrawn. Decompression and drainage could then be carried out following a new puncture guided by the demonstrated intrahepatic bile duct anatomy.

Percutaneous transhepatic intubation of bile ducts by drainage catheter

Decompression and drainage of the bile duct system were performed with the technique described by HOEVELS et coll (1978b,c). The opacified duct system was punctured from the right midaxillary line with a 25-cm stylet

fitted with a polyethylene catheter (ID/OD 1.0/1.6 mm). Guided by biplane fluoroscopy, a peripheral bile duct of the right liver lobe was punctured, achieving a long intraluminal course of the drainage catheter. This will reduce the risk of damage to larger arteries and dislodgement of the catheter by respiratory movements. When the tip of the puncture catheter was positioned within a bile duct, a 0.9 mm guide wire with a slightly curved, soft tip was introduced and manipulated towards the site of the obstruction (Fig. 4). If the occlusion was not complete, the guide wire was passed through the stenosed part into the distal part of the common bile duct or the duodenum (Fig. 5). The puncture catheter was removed and exchanged for the final drainage catheter (ID/OD 1.4/2.2 mm) which was manipulated over a specially designed hard guide wire with a 10-cm flexible segment at its tip (LUNDERQUIST et coll 1979) (Surgimed, Denmark) through the obstruction (Fig. 6). Internal bile drainage was made possible by multiple side-holes proximally and distally to the site of the obstruction. The holes must be placed according to the pathologic findings in the individual case. Taking care to avoid any kinking, the catheter was sutured to the skin and connected to a collecting bag.

Occasionally the obstruction could not be passed by the guide wire or the drainage catheter on the first trial. After two or three days of external decompression and daily irrigation with saline, the amount of bile inspissation, cell detritus, and edema of the wall of the duct was reduced and the possibility of passing the obstruction with the drainage catheter was markedly increased.

When a satisfactory position of the catheter was obtained, plain films of the right upper quadrant were taken daily during the first week after intubation to record possible catheter displacement. Dislodgement of the catheter was most frequent during the first days. If noted early the position of the catheter could easily be readjusted.

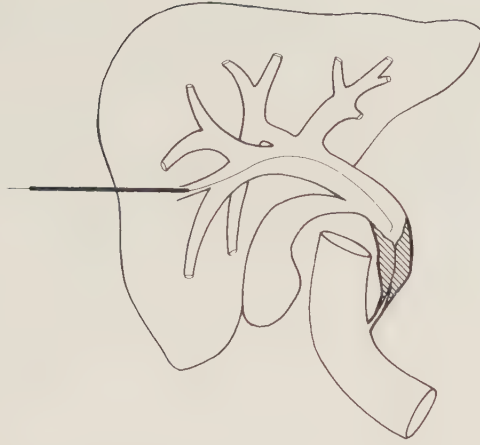


Fig. 4. Puncture catheter with tip in intrahepatic bile duct. Guide wire inserted through catheter with slightly curved tip proximally to tumor obstruction of extrahepatic bile ducts.

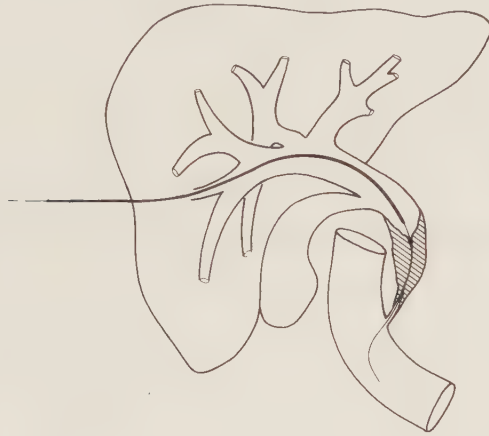


Fig. 5. Puncture catheter advanced into extrahepatic bile ducts. Guide wire passed through obstructed bile duct segment with tip in duodenum.

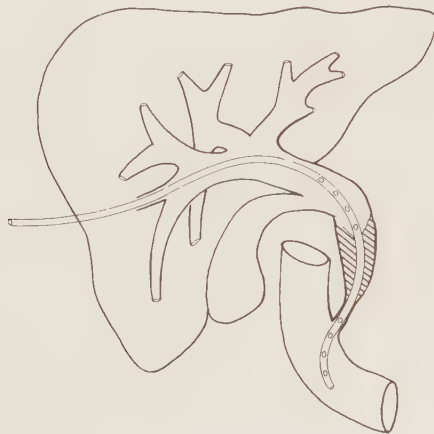


Fig. 6. Drainage catheter passed through tumor obstruction. Side-holes for internal drainage proximally and distally to site of obstruction.

Regardless of the amount of external bile drainage, the catheter was flushed with saline twice each day. The catheter was blocked when the amount of bile drained externally had decreased below 100 ml/24 h and it could be assumed that the internal drainage was functioning well. Serum alkaline phosphatase and serum bilirubin were checked regularly for early detection of cholestasis indicating insufficient internal drainage by the indwelling catheter.

Angiography

The celiac and superior mesenteric arteries were examined simultaneously in 14 patients through injection of 40 ml contrast medium (370 mg I/ml) at a flow rate of 10 ml/s. For better visualization of the superior mesenteric and portal veins, 25 mg of tolazolin hydrochloride diluted in 10 ml saline was injected into the superior mesenteric artery prior to the injection of 60 ml contrast medium (370 mg I/ml) at a flow rate of 12 ml/s. The hepatic artery was examined selectively in 10 patients (30 ml contrast medium, 370 mg I/ml, flow rate 8 ml/s) including infusion hepatic angiography (50 - 60 ml contrast medium, 370 mg I/ml, flow rate 2 - 3 ml/s). The gastroduodenal artery was studied in four patients through injection of 15 - 20 ml contrast medium (370 mg I/ml) at a flow rate of 4 - 6 ml/s.

RESULTS

Post mortem studies

The anatomic relationship of the biliary ducts and portal vein was analyzed in three separate divisions:

- 1/ Porta hepatis - The confluence of the right and left hepatic duct was anterior to the bifurcation of the portal vein in seven and anterior to the right main branch of the portal vein in 18 of 25 cases. In no case was the junction of the hepatic ducts located anteriorly to the left main portal vein branch. The portal vein or its right main branch were in direct contact with the junction of the hepatic ducts in all cases.
- 2/ Hepatoduodenal ligament - Within the approximately 3 to 4-cm passage through the lesser omentum, the portal vein kept in close contact with the common bile duct in the majority of cases. The portal vein was somewhat medial and posterior to the biliary ducts. There was direct contact between these two structures in seven cases and a distance not exceeding 0.5 cm in 14. In four cases, however, the common bile duct was separated from the portal vein by an interspace of 1 cm.
- 3/ Duodeno-pancreatic region - When approaching the duodenum the common bile duct (retroduodenal part) deviated gradually from the portal vein, which continued to descend in an oblique (=inferior-medial) direction, whereas the duct followed a more inferiorly directed course. The distance between the two structures ranged between 0.5 and 1.5 cm. In the majority of cases the common bile duct descended 1.0 cm to the right of the portal vein, which was in a slightly posterior position in relation to the duct. The

most medially located part of the extrahepatic bile duct system was in all cases shown to be the region of transition between the retroduodenal and pancreatic sections. Further on, inferiorly, the common bile duct deviated most often in a gentle curve towards the right before reaching its final (=intramural) segment. The distance between the pancreatic part of the common bile duct and the distal part of the superior mesenteric vein, i.e. the largest splanchnic vein at this level, ranged between 1.5 and 3.5 cm. In 20 of 25 cases this interspace measured between 2.5 and 3.0 cm. There was not cases of ductal or portal venous anomaly in this study.

Clinical studies

All tumors in the 16 patients were adenocarcinomas. At operation and/or autopsy the tumor was found to be localized proximal to or at the main hepatic duct junction in six patients, at the junction of the cystic and common hepatic ducts in two, and in the retroduodenal and pancreatic segments of the common bile duct in three. The tumor appeared to involve the gallbladder and the proximal segment of the extrahepatic ducts in three cases, whereas in one patient it appeared to affect the gallbladder and the distal segments of the extrahepatic bile ducts (II:Fig. 1, Table 1).

The site of the bile duct occlusion was demonstrated at PTC in all patients. The bile duct system of the right lobe of the liver was not completely shown in four of seven cases with tumors at the main hepatic duct junction(six cases) and in the right hepatic duct (one case). The actual extent of tumor involvement of the intrahepatic ducts was underestimated in all these seven patients. The bile ducts of the left lobe of the liver were not filled, or incompletely filled with contrast medium in five of seven cases when the tumor was localized proximal to or at the main hepatic duct junction. A separate puncture of the left biliary duct system was performed in two patients, giving additional diagnostic information. In all nine patients with the tumors

located distal to the hepatic confluence, the bile ducts in both lobes of the liver were adequately filled with contrast medium.

The arteriographic examinations were non-conclusive even in retrospect as to the existence of a tumor in seven of a total of 14 patients. The approximate tumor size in these patients ranged between 2 x 2 cm and 4 x 5 cm. In two of these cases the celiac and superior mesenteric arteries were catheterized. In five patients, in addition, the common hepatic artery was catheterized.

Positive findings at arteriography were observed in the remaining seven patients. Single vessels strongly suggesting neoplastic origin or tumor-infiltrated arteries were demonstrated in four patients close to the bile duct stricture shown at PTC or in the area of the gallbladder. Tumor encasement of the hepatic arteries close to the hilum of the liver was found in two patients; one of these patients had encasement of the gastroduodenal artery, in addition. Multiple neoplastic vessels were demonstrated in the central part of the liver close to the porta hepatis in one patient. In the latter three patients partial compression/infiltration of the portal vein was observed during the venous phase of superior mesenteric angiography. No venous tumor changes were demonstrated in five patients with good visualization of the portal vein. In six cases no conclusion as to detailed morphology of the portal vein was possible because of low concentration of the contrast medium (Table 1).

At PTP the confluence of the portal vein, its main stem, and the proximal segment of its intrahepatic branches were distinctly demonstrated in all 16 patients. No pathologic findings were shown in seven cases.

Positive findings at PTP were observed in the remaining nine patients. Partial compression, or infiltration/occlusion of the main portal vein branch to the right or left lobe was shown in three patients in whom the

TABLE 1. Correlation of findings at PTP with those at venous phase of celiac and superior mesenteric angiography.

P T P	Venous phase of angiography	Tumor compression/ infiltration of veins	Normal findings
	Tumor compression/ infiltration of veins	3	4
	Normal findings	0	7 ^{x/}

In two additional patients only PTP was performed. Invasion of portal vein branches was observed in both cases.

x/ In six of these patients no conclusion as to detailed morphology of the portal vein was possible at angiography because of low concentration of the contrast medium in the venous phase.

tumor was located at the confluence of the hepatic ducts. Compression/infiltration of the portal vein branch to the ventrocranial or dorsocaudal segments of the right lobe was present in three patients in whom the tumor was located close to the confluence of the hepatic ducts. Compression/infiltration of the main stem of the portal vein was demonstrated in three patients in whom the tumor was located distally to the confluence of the hepatic ducts.

Correlations of findings at surgery with those at angiography and PTP

In three of 14 patients in whom angiography was performed, tumor encasement of the hepatic arteries adjacent to and in the porta hepatis was observed. Compression and infiltration of the portal vein was demonstrated at indirect (=arterial) portography and confirmed at PTP in these cases. At operation and/or autopsy, extensive tumor growth was found at the hilum of the liver and in the hepatoduodenal ligament. In four patients in whom arteriography showed fine neoplastic vessels and/or tumor-encased arteries, PTP was normal in two patients, whereas portal vein infiltration was present in the remaining two patients. Extensive surgery was performed in two of these patients who had an approximately 2.5 x 3.5 cm tumor. Microscopy disclosed in both cases that the tumor was not totally removed. In the other two patients a bile duct endoprosthesis was operatively inserted as a palliative measure because of non-resectability of the tumors. PTP was normal in one of the latter patients and demonstrated compression of the portal vein branch to the dorsocaudal segment of the right liver lobe in the remaining patient.

In the seven patients in whom angiography did not demonstrate tumor signs, PTP was normal in five. In two patients, PTP gave hints of infiltration of the liver by an approximately 2 x 3 cm tumor. One of these patients is alive and without symptoms of tumor recurrence four years after right hemi-

hepatectomy and construction of an anastomosis between the left hepatic and common bile ducts. In the remaining patients the tumor was not radically resected at operation. The approximate tumor size interpreted at operation ranged between 2 x 3 cm and 4 x 5 cm in these six cases.

DISCUSSION

The extrahepatic bile ducts and the portal vein are in close association in the porta hepatis and the hepatoduodenal ligament. As demonstrated by the present topographic study, the bifurcation of the portal vein is situated in direct contact with the superimposing junction of the hepatic ducts and the distance between the portal vein and the extrahepatic bile ducts does not exceed one cm throughout their course in the hepatoduodenal ligament.

The relationship between the portal vein and the biliary ducts in the central part of the liver, the porta hepatis and the hepatoduodenal ligament therefore strongly suggests that a cholangiocarcinoma may have involved the portal venous system at the time when surgical procedures were considered.

Carcinomas of the proximal bile ducts are rarely resectable. The most favorable results have been reported in patients with carcinoma of the distal common bile duct and the ampulla of Vater (WARREN et coll 1975). Tumor infiltration of the hepatic artery and/or portal vein frequently excludes curative resection (THORBJARNARSON 1975). GLENN and HAYS (1954) found in two-thirds of cases with malignant tumors of the extrahepatic biliary tract at autopsy "that an apparent complete removal of the tumor tissue would not have been difficult if both the hepatic artery and the portal vein had been expendable". They concluded in 1954 "that some form of vascular graft will ultimately make such resection a possibility". FORTNER et coll in 1974 reported resection of involved vascular structures along with the primary tumor, but for bile duct carcinoma this has met with little success, most patients dying in the postoperative period.

Surgical intervention may benefit essentially from a thorough preoperative radiological evaluation of a patient with extrahepatic cholestasis suspected to be caused by a malignant tumor. The site of the bile duct obstruction and the anatomy of the intrahepatic biliary system can be evaluated by PTC. The diagnostic significance of PTC was markedly increased in combination with percutaneous fine-needle aspiration biopsy, which made possible a cytological diagnosis of the obstructive lesion of the bile ducts in nine of 16 cases. No conclusion, however, should be drawn from the findings at PTC as to the extension of a tumor lesion outside the ducts. A short tumor occlusion/stricture of the extrahepatic bile ducts may give a false impression of tumor size and extirpability. In one case presented in this material a 1-cm stricture of the common hepatic duct was combined with extensive tumor infiltration of the portal vein. The bile ducts distally to the tumor lesion may be visualized either by ERC or PTC whenever a catheter can be manipulated through the tumor stricture. Subsequent preoperative bile duct decompression and combined internal/external bile drainage through the percutaneous transhepatic catheter may ameliorate liver function and the general condition of the patient prior to palliative or radical surgical procedures.

Various authors (REUTER et coll 1971, KAUDE and RIAN 1971, HAERTEL and ANDERSON 1976, WALTER et coll 1976) have investigated the diagnostic effectiveness of angiography in patients with carcinoma of the extrahepatic bile ducts and found a high diagnostic yield. This contrasts markedly with the results of the present investigation which are in line with the findings reported by SPRAYREGEN and MESSINGER (1974). We observed no pathological findings at angiography in seven of 14 patients with carcinoma of the extrahepatic biliary tract. At subsequent operation in six of the patients, a non-resectable carcinoma was found. In one case the tumor was resected and the patient is free of recurrence four years after operation. In the remaining seven patients angiography indicated non-extirpability in three. At operation, however, in none of the seven cases could the tumor be removed radically.

SPRAYREGEN and MESSINGER (1974) performed selective examinations of the celiac and superior mesenteric arteries in patients with bile duct carcinoma and explained the higher percentage of tumor signs by a more selective mode of examination applied by other authors (REUTER et coll 1971, KAUDE and RIAN 1971, HAERTEL and ANDERSON 1976). In addition to examination of the celiac and superior mesenteric arteries we performed selective examinations of the common/proper hepatic arteries in 10 patients and of the gastroduodenal artery in four. This resulted in a very poor diagnostic yield. The number of patients in the present study is too small to permit conclusions on the value of angiography in bile duct carcinoma. There seems, however, to be little reason to perform superselective studies in order to demonstrate the extent and resectability of bile duct carcinoma.

The low incidence of portal vein involvement in carcinoma of the extrahepatic bile ducts is pointed out in several reports (BOIJSEN and REUTER 1967, REUTER et coll 1971, KAUDE and RIAN 1971, HAERTEL and ANDERSON 1976, WALTER et coll 1976). This conclusion is drawn from the indirect (=arterial) portogram. From the present studies (I + II) it is evident that tumor involvement of the portal vein occurs in most cases and then is underestimated when this conclusion is based on the venous phases of celiac/superior mesenteric studies. Tumor affection of the portal venous system was observed by PTP in totally nine of 16 patients reported herein. In seven of these nine patients arteriography was performed and revealed tumor infiltration/compression of the portal vein in three cases only. In addition, PTP indicated non-extirpability in one of these patients because of infiltration/compression of the main stem of the portal vein and gave hints of infiltration of the liver in five patients because of intra- or extra-hepatic invasion of the portal vein.

There was strong evidence that compression of the portal vein by a bile duct carcinoma may be equivalent to malignant infiltration of the vessel. In six patients mild-to-marked compression of different segments of the portal venous system at PTP was found to represent tumor infiltration of the vessels at surgery - autopsy and/or microscopy. This is in line with

the findings of NORTON and EISEMAN (1975) who resected the portal vein which was surrounded but not penetrated by a pancreatic carcinoma in three patients. Microscopy revealed tumor infiltration of the wall of the vein in all cases. RÖSCH (1959), RÖSCH and HERFORT (1962), MILLER et coll (1966), and SATO et coll (1967) reported similar results from studies of the contribution of splenoportography in biliary and pancreatic malignancy. If the main stem of the portal or splenic vein was compressed by the malignant tumor originating from the biliary/pancreatic region, the tumor was regularly found to infiltrate the vein and deemed non-resectable at operation.

Infiltration of intrahepatic portal vein branches indicates invasion of the liver by the bile duct carcinoma. This apparently does not necessarily exclude curative surgery, as proven in one patient from this study, provided that the main stem of the portal vein is free of tumor growth. In all patients, however, in whom angiography and/or PTP demonstrated tumor involvement of the extrahepatic portal venous system, the lesion was not radically extirpable. Finally, there is evidence from one case in this study that even PTP, which is superior to arterial (=indirect) portography because of the higher concentration of contrast medium in the portal vein, may fail to demonstrate existing tumor growth into the wall of the portal vein (II:table 1: case 15). Furthermore, at operation tumors were regarded as non-extirpable in seven patients where neither arteriography nor PTP gave this essential information. This part may be due to the fact that the extrahepatic bile ducts run anterior to the portal vein and its branches in the porta hepatis and the hepatoduodenal ligament. PTP in lateral projection may give additional information. In this material PTP was performed in the lateral projection in four patients in whom no tumor involvement of the main stem of the portal vein was found at operation. One of these patients had tumor invasion of an intrahepatic portal vein branch, which was better shown in anterior-posterior projection.

TABLE 2.

Case No.	Age/Sex	Diagnosis
1	58 M	Carcinoma of body and tail of pancreas, metastasis in porta hepatis
2	62 F	Carcinoma of gallbladder, metastases in porta hepatis and hepatoduodenal ligament
3	89 F	Carcinoma of gallbladder, infiltration of hepatoduodenal ligament
4	83 F	Carcinoma of distal segment of common hepatic/proximal segment of common bile duct
5	73 M	Carcinoma of gallbladder, infiltration of porta hepatis and hepatoduodenal ligament
6	53 F	Periampullary carcinoma, multiple liver metastases
7	22 F	Crohn's disease, chronic inflammatory mass in porta hepatis and hepatoduodenal ligament
8	70 M	Periampullary carcinoma
9	77 F	Carcinoma of proximal segment of common bile duct
10	88 M	Periampullary carcinoma
11	90 F	Periampullary carcinoma
12	64 F	Carcinoma of breast, metastasis in hepatoduodenal ligament
13	68 M	Periampullary carcinoma, multiple liver metastases

Non-surgical endoprosthesis in obstructive lesions of extrahepatic bile ducts (III)

MATERIAL AND METHODS

In 12 patients a malignant tumor and in one an inflammatory mass (Crohn's disease) in the porta hepatis and the hepatoduodenal ligament obstructed the extrahepatic bile ducts (Table 2). The platelet count was above $50,000/\text{mm}^3$ and the APTT (Activated Partial Thromboplastine Time) below 35 seconds in all patients when the endoprosthesis was inserted.

Insertion of bile duct endoprosthesis

As endoprosthesis a teflon tube (ID/OD 3.0/4.0 mm, William Cook ApS, Denmark) was used. The proximal end of the tube was slightly flared, giving an outer diameter varying between 5 and 6 mm. The distal end was slightly tapered. The length and shape of the endoprosthesis were chosen according to the site of the bile duct stricture/occlusion in the individual case. The procedure was performed with local anesthesia.

The passage of the bile duct stricture/occlusion by a guide wire and catheter was the prerequisite for the percutaneous transhepatic insertion of a bile duct endoprosthesis. The technique of percutaneous transhepatic biliary tract intubation is described on page 21.

When the endoprosthesis was to be introduced a specially designed hard guide wire (Surgimed, Denmark) with a 10-cm flexible segment at its tip was introduced into the indwelling drainage catheter until the tip of the guide wire reached the duodenum. Following local anesthesia around the puncture canal in the skin, subcutis and intercostal muscles, the drain-

age catheter was removed with the guide wire tip remaining in the duodenum. The bile duct stricture/occlusion was widened by introduction of two coaxial dilatation teflon catheters. Catheter I (ID/OD 1.8/2.7 mm) was introduced over the guide wire until its tip was in the duodenum. Catheter II (ID/OD 3.0/4.0 mm) was introduced over Catheter I (Fig. 7). During this maneuver attention must be paid that Catheter I is not pushed further into the duodenum. When the tips of both catheters were in end-to-end position in the duodenum, Catheter II was removed and re-inserted a few times. By these means a canal through the liver parenchyma was established, facilitating the insertion of the endoprosthesis. Catheter II was then removed. The endoprosthesis was mounted on Catheter I which remained with its tip in the duodenum. The endoprosthesis was moved by hand, gliding over Catheter I until its proximal (=flared) opening reached the skin of the patient. Thereupon Catheter II was remounted on Catheter I behind the endoprosthesis and applied to push the endoprosthesis into the intended position (Fig. 8). The maneuver was performed under fluoroscopic control. Care was taken not to place the endoprosthesis further distally into the bile duct than planned. Catheters I and II were removed with the special guide wire still in place in the duodenum. The procedure was terminated by inserting a polyethylene catheter (ID/OD 1.4/2.2 mm) over the guide wire with its tapered tip in the endoprosthesis. The guide wire was removed and the catheter fixed to the skin by a suture. The latter catheter was finally removed after a few days when adequate drainage via the endoprosthesis was demonstrated by injection of contrast medium. In cases with non-functioning endoprosthesis the catheter was left in place for external bile drainage.

In patients with an endoprosthesis the total serum bilirubin and serum alkaline phosphatase levels were regarded as parameters of bile drainage effectiveness. In patients with a permanent bile duct endoprosthesis and a percutaneous transhepatic catheter, the amount of externally drained bile was taken into consideration.

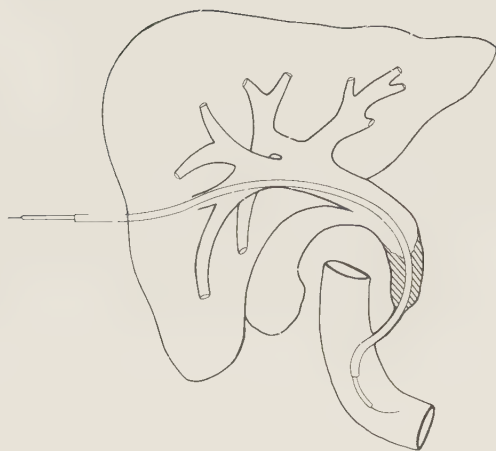


Fig. 7. Catheters I and II inserted coaxially over guide wire through tumor stricture with tip in duodenum.

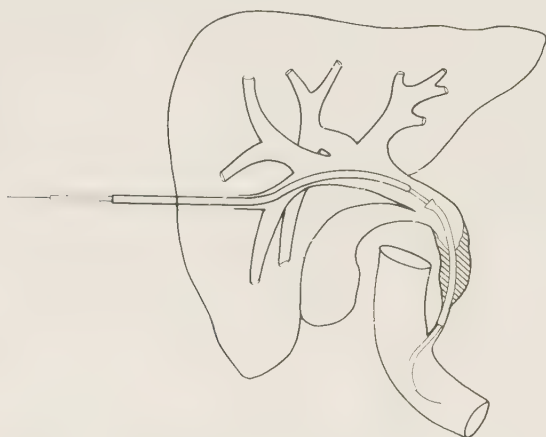


Fig. 8. Endoprosthesis and Catheter II advanced over Catheter I. Endoprosthesis in region of tumor stricture.

RESULTS

The site of the bile duct obstruction in the 13 patients of this study was localized by PTC in the common bile duct in eight cases, in the common hepatic duct in two, and in the main junction of the hepatic ducts in two. One patient had a tumor stricture of the main junction of the hepatic ducts and in addition, a tumor stricture of the common bile duct (Table 2). The length of the endoprosthesis varied between four and 12 cm (mean 8 cm). The position and length of the endoprosthesis in relation to the length of the bile duct stricture/occlusion appear in Table 3. The proximal orifice of the endoprosthesis was placed in the right hepatic duct in four patients, whereof three had tumor occlusion of the main junction of the hepatic ducts. The duct system of the left lobe of the liver was not drained in these cases.

The percutaneous transhepatic catheter which was used for flushing the endoprosthesis and controlling its patency was removed in seven patients after four days to three weeks. Subsequently the bile was drained via the endoprosthesis only during a period ranging from one to 35 weeks. In no case were the total serum bilirubin and serum alkaline phosphatase reduced to normal values (II:Table 1). Six patients had a permanent transhepatic catheter because of insufficient bile drainage through the endoprosthesis. In three of these cases the endoprosthesis was introduced in a position with either the distal or the proximal orifice pointing against the wall of the bile duct. In one case the endoprosthesis was inadvertently occluded by Bucrylat. Spontaneous dislocation hindered bile flow through the endoprosthesis in two cases. The value of total serum bilirubin and serum alkaline phosphatase during the drainage period and the volume of bile drained during 24 hours through the percutaneous transhepatic catheter in this group are shown in III:Table 2.

TABLE 3

Case No.	Site of bile duct stricture/ occlusion at PTC	Appr. length of bile duct stricture/ occlusion	Length and location of bile duct endoprosthesis
1	Confluence of hepatic ducts	4 cm	12 cm, <u>right</u> hepatic duct to distal segment of common bile duct
2	Confluence of hepatic duct(s) and proximal segment of common bile duct	2 cm 2.5 cm	10 cm, <u>right</u> hepatic duct to distal segment of common bile duct
3	Confluence of hepatic ducts	5 cm	8 cm, <u>right</u> hepatic duct to distal segment of common bile duct
4	Common hepatic duct	4 cm	8 cm, common hepatic duct to distal segment of common bile duct
5	Common hepatic duct	4 cm	11 cm, common hepatic duct to duodenum (2cm beyond papilla Vateri)
6	Distal segment of common hepatic duct	6 cm	10 cm, common hepatic duct to distal segment of common bile duct
7	Distal segment of common bile duct	4 cm	7 cm, common bile duct to papilla Vateri
8	Proximal segment of common bile duct	5 cm	9 cm, common hepatic duct to duodenum (2 cm beyond papilla Vateri)
9	Proximal segment of common bile duct	4 cm	8 cm, common hepatic duct to papilla Vateri
10	Distal segment of common bile duct	3 cm	5 cm, common bile duct to papilla Vateri
11	Distal segment of common bile duct	4 cm	7 cm, common hepatic duct to papilla Vateri
12	Distal segment of common bile duct	3 cm	11 cm, <u>right</u> hepatic duct to duodenum (1 cm beyond papilla Vateri)
13	Distal segment of common bile duct	3 cm	8 cm, common hepatic duct to papilla Vateri

Complications

Four patients developed clinical signs of cholangitis one to three days after insertion of the endoprosthesis. Septicemia was verified by blood cultures in one of these patients. Incorrect placement of the endoprosthesis caused functional occlusion of either its proximal or distal orifice by the wall of the bile ducts in three patients. Spontaneous dislocation of the endoprosthesis resulted in ineffective bile drainage in two whereas minor dislodgement (i.e. < 2 cm) did not seem to influence the bile passage through the teflon tube in three cases.

The formation of an intrahepatic aneurysm adjacent to the puncture canal in the right lobe of the liver occurred in one patient who developed severe hemorrhages. In order to stop the otherwise uncontrollable bleeding, therapeutic embolization of the aneurysm was performed. Besides partial occlusion of the aneurysm, embolization of the majority of the segmental arteries of the liver was accomplished. The hemorrhage ceased after this procedure. Three days later the patient developed liver failure and died. Transitory formation of blood clots in the hepatic ducts in connection with the bile duct intubation was observed in one patient. No clinical signs of hemobilia were noticed in this patient.

Findings at autopsy

Postmortem examination was performed in six of nine patients who had a bile duct endoprosthesis for a period of between two and 24 weeks prior to death. The endoprosthesis was patent in four cases after two, three, three, and eight weeks, respectively. The bile ducts were slightly (three cases) or moderately (one case) dilated. In one patient with marked hemobilia because of severe hemorrhage from an intrahepatic aneurysm, the endoprosthesis was completely occluded by blood clots. Complete obliteration of the endoprosthesis by inspissated bile and cell de-

bris was found in one patient five months after insertion of the teflon tube. Suppurative cholangitis was present in two patients. In one of these the endoprosthesis drained the right liver lobe only. The ducts of the left liver lobe were grossly dilated and contained purulent bile. The other patient had multiple abscess formations combined with purulent cholangitis in both liver lobes.

DISCUSSION

When jaundice is caused by extrahepatic cholestasis because of a malignant tumor, death will result in the majority of cases from hepatic failure due to progressive biliary obstruction rather than from massive neoplastic invasion of the liver or tumor spread to extrahepatic sites. It is therefore essential to establish effective bile duct decompression in these cases as soon as the final diagnosis has been established. Palliative relief of biliary obstruction by non-surgical procedures must be considered whenever the age and general condition of the patient and/or extensive tumor growth as well as the site of the tumor preclude operative bile duct decompression. In recent years considerable experience with palliative bile drainage through percutaneous transhepatic catheters has been collected in various institutions (GLENN et coll 1962, AHNLUND and MORALES 1963, MARIONS and WIECHEL 1964, KAUDE et coll 1969, LANG 1974, MOLNAR and STOCKUM 1974, TAKADA et coll 1976, MORI et coll 1977, TYLÉN et coll 1977, ROSENMAN et coll 1977, ARIYAMA et coll 1978, BURCHARTH and NIELBO 1976, HENRY et coll 1978, HOEVELS et coll 1978 b,c, MOLNAR and STOCKUM 1978, NAKAYAMA et coll 1978, PROBST et coll 1978, RING et coll 1978, GÜNTHER et coll 1979, HANSSON et coll 1979). This method has proved effective in accomplishing relief of biliary obstruction by external drainage or by combined internal/external drainage following bile duct intubation by a percutaneous transhepatic catheter. There are, however, some shortcomings involved in these methods. The drainage may be interfered by spontaneous catheter dislodgement and/or infection of the biliary duct system through the external catheter (HOEVELS et coll 1978 b, c, HANSSON et coll 1979). External bile leakage around the percutaneous transhepatic catheter and inflammation and pain at the puncture site may be further disadvantages of the procedure. Furthermore, the knowledge that the catheter and bag must be borne permanently constitutes a severe psychological strain for the patient. These obvious disadvantages of bile drainage via percutaneous transhepatic catheter strongly motivated us to test the efficacy of the bile duct endoprosthesis.

We applied a large-bore teflon tube in order to obtain sufficient bile flow through the endoprosthesis. The proximal orifice of the endoprosthesis was expanded to reduce the risk of dislodgement in distal direction. The teflon tube was designed without side-holes, to minimize the risk of occlusion by debris material from the adjacent wall of the bile duct. The insertion of the dilatation catheter and the endoprosthesis was greatly facilitated by the hard guide wire. Kinking of the catheters did not occur and the endoprosthesis was brought into final position without major problems.

The functioning of the endoprosthesis was regarded as partially effective in seven of 13 patients. The serum bilirubin and serum alkaline phosphatase declined to normal values in none of these cases. In six cases bile drainage through the endoprosthesis was ineffective and depended exclusively on the percutaneous transhepatic catheter. Malfunction was due mainly to primarily improper position or dislodgement of the endoprosthesis. Neither BURCHARTH (1979) nor PEREIRAS et coll (1978) observed impaired drainage function because of unfavorable position of the endoprosthesis. Dislodgement of the tube was reported by BURCHARTH in only one of 41 patients. PEREIRAS et coll did not report dislodgement of the endoprosthesis in any of 12 patients. Furthermore, contrary to our results, BURCHARTH (1978) achieved satisfactory drainage function in 65 per cent of patients with a thin polyethylene endoprosthesis (ID/OD 1.5/2.0 mm). PEREIRAS et coll (1978) reported the disappearance of jaundice in 10 of 12 patients who had the same type of endoprosthesis (ID/OD 3.0/4.0 mm) as that used in the present study. Optimal bile drainage through the endoprosthesis should result in normalization of the serum bilirubin and alkaline phosphatase levels, if cholestasis is caused by extrahepatic bile duct occlusion only. Technical shortcomings such as the design of the endoprosthesis and/or its final position in the extrahepatic bile ducts must therefore be considered the main reason for functional disorder. BURCHARTH (1978) and BURCHARTH et coll (1979) used a polyethylene tube with side-holes and did not observe occlusion of the endoprosthesis in any case.

It is evident from the present study that the risk of cholangitis is correlated to the length of the drainage period through a percutaneous transhepatic catheter. This confirms the observation of HANSSON et coll(1979) who reported cholangitis in about 25 per cent of patients with a percutaneous transhepatic drainage catheter. Apparently the frequency of bile duct infection can be reduced markedly when the endoprosthesis is introduced immediately following the diagnostic PTC (BURCHARTH 1978, BURCHARTH et coll 1979).

The formation of aneurysms of intrahepatic arteries following PTC and needle biopsy of the liver is on record (WALLACE et coll 1972, HELLEKANT 1976, MOSKOWITZ et coll 1978). We experienced severe hemorrhaging in one patient in whom angiography prior to and following the percutaneous transhepatic insertion of a drainage catheter and an endoprosthesis demonstrated the formation of an intrahepatic aneurysm adjacent to the puncture tract. To reduce the risk of vascular damage it is therefore essential to catheterize a peripheral bile duct of the right liver lobe when the puncture is to be performed from the right side of the patient. By these means the drainage catheter and the endoprosthesis pass the liver parenchyma intraductally to the greatest possible extent and the risk of mechanical damage to the arteries of the liver is diminished.

From the experience gained from the present studies and those of BURCHARTH (1978) and BURCHARTH et coll (1979), it seems evident that sideholes in both the proximal and distal part of the endoprosthesis are crucial for sufficient drainage function. Complete occlusion of the tube by the wall of the bile duct may thus be avoided. Fine tubes have not proved to be less traumatic than large-bore tubes, which rationally should provide better bile drainage and imply less risk of occlusion by cell material and inspissated bile.

Percutaneous transhepatic portography and infusion angiography in malignant liver tumors (IV)

MATERIAL AND METHODS

The diagnostic yield of percutaneous transhepatic portography was compared with that of infusion hepatic angiography in 21 patients with primary (six cases) and metastatic (15 cases) lesions of the liver.

The technique of PTP was described on page 16 . The technical quality of PTP was judged for each lobe as good, moderate, or poor according to the following criteria:

1. visualization of the intrahepatic portal vein branches,
2. accumulation of contrast medium in the liver parenchyma.

PTP was classified as more, equally, or less informative than infusion hepatic angiography in visualizing space-occupying lesions. In positive findings at PTP, the portal venous and parenchymatous (=sinusoidal) phases were evaluated separately according to their diagnostic information. PTP was regarded as non-contributory to the diagnosis when it demonstrated tumor changes to a lesser degree than or to the same degree as infusion hepatic angiography.

Hepatic infusion angiography

For the infusion studies the catheter was placed in the common hepatic artery in 12 cases and in the proper hepatic artery in seven. In two cases catheterization was carried out of the right hepatic artery which originat-

ed from the superior mesenteric artery. In the examination of the left liver lobe in these two cases infusion hepatic angiography was not performed. The left gastric artery contributed in no case to the blood supply of the left lobe of the liver. The infusion studies were performed with the patient supine and an injection of 50 - 60 ml contrast medium (370 mg I/ml) at a flow rate of 2 - 4 ml/sec. The quantity of contrast medium and the flow rate were determined by estimating the degree of hepatic arterial blood flow and the size of the liver. In addition, care was taken to minimize the risk of contrast medium reflux into the splenic artery. Filming for infusion angiography was spread over 32 seconds exposing 16 films at 2-second intervals. Only cases where the hepatic infusion study was regarded as technically optimal are included in the study.

RESULTS

At infusion hepatic angiography tumor(s) were found in both lobes in 10 cases, in the right lobe only in seven, and in the left lobe only in four. In 11 patients multiple tumors were present, whereas in 10 a solitary tumor was found. Four solitary tumors were localized in the right lobe and four in the left. In two cases a solitary lesion involved both liver lobes. The tumors were angiographically hypervascular in 15 cases and hypovascular in six. The technical quality of PTP in the right liver lobe was good throughout. When the contrast medium was injected into the main stem of the portal vein the visualization of the portal vein branches and parenchyma of the left liver lobe was classified as good in 10 cases and poor in 11. In six cases the left liver lobe was selectively investigated by injection of contrast medium into the left main branch of the portal vein, resulting in good visualization of this lobe. PTP revealed in 14 of 21 patients tumors which had been detected by infusion hepatic angiography. They were found in both lobes in six cases, in the right lobe only in six, and in the left lobe only in two. In eight of 12 cases with tumors of the right lobe of the liver, the essential portographic diagnostic information was obtained from the portal venous phase of PTP, and in four cases from the sinusoidal phase. Tumors of the left lobe of the liver

were detected in five of eight cases in the portal venous phase and in three cases in the sinusoidal phase.

The detectability of space-occupying lesions by PTP was dependent on their size, number and location. Whereas all tumors beyond the size of 7 cm and most multiple metastases (1 - 2 cm) were demonstrated, solitary lesions smaller than 2 cm located in the central part of the liver were not detected. In tumor lesions of the right liver lobe, PTP provided approximately the same diagnostic information as infusion hepatic angiography in six of 17 cases. In six cases the number of metastases was shown to a lesser degree. In five cases PTP resulted in falsely negative findings.

In tumor lesions of the left lobe, PTP in three cases provided the same diagnostic information as infusion hepatic angiography regarding the extent of the space-occupying lesion. In one case PTP showed multiple lesions in the left lobe which were not demonstrated by infusion hepatic angiography. In seven cases PTP resulted in falsely negative findings. In four cases the tumor size was better delineated at hepatic angiography.

DISCUSSION

For the evaluation of the diagnostic effectiveness of PTP in malignant lesions of the liver, access to a reliable reference method is crucial. Furthermore, both diagnostic methods must be performed within a very short period of time to exclude alteration of the underlying liver pathology. It was regarded rational to perform PTP shortly after the verification of malignant lesions of the liver by infusion hepatic angiography.

According to the results of postmortem injection studies, the blood supply of malignant tumors of the liver comes only from the hepatic arteries (SEGALL 1923, BREEDIS and YOUNG 1954, HEALEY 1965, PINET et coll 1972). Visualization of the intrahepatic portal venous system will therefore demonstrate only indirect signs of expansive lesions, rendering impossible differentiation between malignant and benign masses.

The diagnostic value of portography depends on the visualization of the complete intrahepatic portal venous system. Due to its ventral position, the portal venous system of the left liver lobe in the majority of cases is insufficiently visualized when the patient is examined supine, regardless of which access to the portal venous system is used. In percutaneous splenoportography this problem may be overcome when the examination is performed with the patient in a prone position. However, the best demonstration of the portal venous system of the left lobe of the liver is obtained through selective catheterization of the left main branch of the portal vein via either the percutaneous transhepatic or the transumbilical access.

In cirrhosis of the liver, obstructive scarring affects predominantly the hepatic veins (HALES et coll 1959, KELLY 1950, PIPER 1961, CARTER et coll 1961). Varying degrees of hepatic vein obliteration may be present in different parts of the liver, resulting in diversion of portal venous

blood from areas with high-flow resistance to areas in which hepatic venous drainage is less obstructed. Permanent or intermittent reversal of blood flow in intrahepatic portal vein branches (HOEVELS et coll 1979 b) will result in non-visualization of large areas of the liver at PTP. Furthermore, distortion and dislodgement of intrahepatic portal vein branches due to destruction and regeneration of liver tissue make the identification of a neoplastic lesion by PTP impossible.

Various authors have studied the value of spleno-, transumbilical-, and peroperative portography in the diagnosis of space-occupying lesions of the liver. Whereas ANACKER et coll (1957) found that only large tumors can be detected by this method, RÖSCH (1959) concluded that even small tumors may be detected in the central part of the right liver lobe. ARONSEN et coll (1969) studied the diagnostic value of preoperative portography in comparison with selective angiography of the celiac artery and found it of value in poorly vascularized tumors of the liver. MATEEV and WIRBATZ (1976) compared the results of transumbilical portography with those of selective examination of the celiac axis and hepatic artery. They found portography to be superior to hepatic angiography. In all previous examinations, however, the results of portography were not compared to hepatic infusion angiography which must be regarded as the most reliable method of liver angiography (WIRTANEN 1973, FREENY et coll 1976).

According to our experience based on the present material, the demonstration at PTP of single tumor lesions as small as 2 - 3 cm seems more likely to be the exception than the rule. Multiple metastases of approximately 1 - 2 cm were seen in most cases, however. Two-to-four-cm metastases in the right liver lobe were detected mainly in the sinusoidal phase of PTP. Overprojection of portal vein branches made the identification of space-occupying lesions of this size impossible in the portal venous phase in all cases but one. Tumors as large as 4 - 5 cm may remain undetected when they are located in the center of the liver engaging both lobes. The true degree of tumor spread in the liver was better demonstrated at infusion hepatic angiography in all cases but one. In this case PTP demonst-

rated multiple lesions of the left lobe of the liver which were not demonstrated by hepatic infusion angiography. Selective catheterization of the left main branch of the portal vein should theoretically overcome the diagnostic problems in lesions of this lobe due to overprojection of the spine and the unfavorable hemodynamics involved with the patient in supine position. However, in the present material it is shown that portography is an unreliable method of detecting solitary lesions smaller than 5 cm in the left liver lobe. Tumor lesions larger than 6 cm in the right liver lobe were visualized at PTP as effectively as at infusion hepatic angiography. Tumors larger than 7 cm in the left liver lobe were easily demonstrated during the portal venous and sinusoidal phases of PTP. In patients with primary liver carcinoma the size of the tumor in most cases was shown better at arteriography. PTP, however, excellently demonstrated the main stem of the portal vein and its intrahepatic ramifications.

GENERAL SUMMARY AND CONCLUSIONS

I + II

The topographic relationship of the portal vein to the extrahepatic bile ducts was investigated by simultaneous portography and cholangiography at postmortem examination in 25 cases. In the region of the porta hepatis the main hepatic duct junction was found to be in direct contact with the portal vein or its branches, whereas minor variations in the interspace between the bile ducts and the portal vein were demonstrated in the hepatoduodenal ligament and the duodeno-pancreatic region. The results of this investigation are discussed with respect to the clinical experience that carcinomas of the extrahepatic bile ducts often involve the proximal part of the extrahepatic biliary system.

From the present studies it appears that PTC is an indispensable method in the demonstration of tumor involvement in carcinoma of the extrahepatic bile ducts. The extent of tumor growth outside the biliary ducts and tumor extirpability were evaluated by angiography and PTP. In the majority of cases both methods were found to be insufficient in providing this essential information. Nevertheless we advocate angiography as indispensable in patients with suspected bile duct carcinoma. When extensive tumor invasion is demonstrated, exploratory laparotomy may be avoided and the patient may instead benefit from the non-surgical methods of decompression and drainage of the biliary system. In normal findings at angiography, complementary PTP may provide information as to tumor extirpability. Normal findings at angiography and PTP do not exclude the presence of a non-extirpable tumor.

III.

A large-bore teflon endoprosthesis was inserted via the percutaneous transhepatic approach in patients with extrahepatic cholestasis. All but one of these patients had a malignant tumor obstructing the extrahepatic bile ducts. We experienced that this method implies the risk of serious intrahepatic lesions. Although this complication cannot be avoided entirely, the application of the correct technique of puncture and catheterization of the intrahepatic bile duct system would seem to be the most essential step in reducing serious vascular damage to the liver arteries. The function of the endoprosthesis was graded as partially effective in only half of the patients and as ineffective in the remaining cases. A major draw-back of the endoprosthesis used herein was the lack of side-holes. Side-holes would seem to be essential in avoiding functional disorder when the proximal and/or distal openings of the endoprosthesis are occluded by the wall of the bile duct. We advocate a large-bore endoprosthesis with side-holes, which rationally should provide better bile drainage than a fine endoprosthesis. Only patients who are not eligible for any kind of surgical bile duct decompression should obtain a non-surgical endoprosthesis.

IV.

The diagnostic value of PTP was evaluated in patients with verified primary and secondary malignant tumors of the liver. The findings at PTP were correlated with those of infusion hepatic angiography, which demonstrated tumor lesions in all cases. PTP was non-contributory to the diagnosis in all but one patient, in whom lesions in both liver lobes were demonstrated at PTP whereas infusion hepatic angiography failed to reveal tumors in the left liver lobe. PTP, like other methods of direct portography, was regarded as obsolete and far inferior when compared to the diagnostic effectiveness of infusion hepatic angiography in primary and metastatic tumors of the liver. This applied to both liver lobes.

PTP provided detailed morphological information of the main stem of the portal vein and its main ramifications. PTP as a complement to hepatic arteriography may give essential information as to the feasibility of partial liver resection rather than aiding in the diagnosis of liver tumor.

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Topographic Relation of Portal Vein to Extrahepatic Bile Ducts. A Combined Portographic-Cholangiographic Study in 25 Cadavers

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12 Figures

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The extrahepatic course of both portal vein and bile ducts and their interrelation are analysed by simultaneous portography and cholangiography in 25 adult cadavers. The findings are correlated with the topographic anatomy in the porta hepatis, the hepatoduodenal ligament, and the duodeno-pancreatic region. Minor variations of the interspace between the bile ducts and the portal vein and superior mesenteric vein are demonstrated. The results of this investigation are discussed with respect to the clinical experience that carcinomas of the proximal part of the extrahepatic bile ducts often engage the bifurcation and the main stem of the portal vein, whereas the confluence of the portal vein and the superior mesenteric vein is more often affected by cancer of the head of the pancreas.

For interpretation of portal venous changes expected to be produced by a cholangiocarcinoma it is important to know the topographic relation between bile ducts and the portal vein at different levels of their extrahepatic course. The extrahepatic part of the biliary duct system and the portal vein come into close association with each other within the porta hepatis and the hepatoduodenal ligament. Tumorous changes affecting the bile ducts in this region may readily spread to the portal vein, whereas lymphatic metastases within the hepatoduodenal ligament may simultaneously influence both the duct system and the veins afferent to the liver. In malignant tumours originating from the head of the pancreas and from structures within the hepatoduodenal ligament or the porta hepatis, surgical intervention may benefit essentially from preoperative evaluation by arteriography, percutaneous transhepatic portography (PTP), and cholangiography (PTC) whether or not radical or palliative operations are performed. PTC cannot provide any information as to tumorous engagement of the large veins adjacent to the extrahepatic biliary ducts. However, due to the predictable anatomical relation of the ductal and venous vascular structures within the porta hepatis and hepatoduodenal ligament, the site of a neoplasm gives a strong hint of the likelihood of tumor growth to the portal vein. It is the purpose of this study to demonstrate the normal anatomical relationship of the ductal and portal venous structures within the porta hepatis, the hepatoduodenal ligament, and the duodeno-pancreatic region by portographic and cholangiographic examinations performed simultaneously postmortem.

Direct and selective access to the portal vein and the biliary system by the percutaneous transhepatic route is by now a well established radiological method. Bile stasis due to malignancy originating from structures of the porta hepatis, the hepatoduodenal ligament, and tumours of the periampullary region represents the main indication for percutaneous transhepatic cholangiography (1, 2, 4, 5, 6, 13, 20, 23, 24, 26, 27, 32, 33, 35, 36, 37, 38, 40, 41,

45, 48, 53, 54, 56). The most common indication for percutaneous transhepatic portography is cirrhosis of the liver with portal hypertension calling for hemodynamic studies of the portal venous system and, in many cases, therapeutic occlusion made possible by the selective access to venous collaterals feeding esophageal varices. Additional indications are suspected neoplasms within the liver, the porta hepatis, the hepatoduodenal ligament, and the pancreas (15, 16, 21, 22, 28, 29, 30, 31, 39, 42, 44, 46, 49, 50, 51, 55, 57, 58).

Material and Method

During routine autopsy of 25 adult cadavers, all of which were fresh hospital specimens, a branch of the superior mesenteric vein within the mesenteric root was dissected and cannulated with a 0.4-cm polyethylene catheter. The tip of the catheter was advanced into the main stem of the portal vein or into one of its main branches. The catheter was fastened with a tight ligature around the cannulated superior mesenteric vein. In 15 cases another 0.2 cm catheter was inserted into the papilla of Vater and secured by a ligature. In 10 cases contrast filling of the biliary tract was achieved by intubation of the gallbladder with a 0.4 cm polyethylene tube. In 15 cases the colon and the small bowel, except for the duodenum, were removed prior to the examination, whereas the abdominal situs was left untouched in 10 cases. Opacification of the intra- and extrahepatic biliary system as well as the cranial part of the superior mesenteric vein, the splenic vein, and the portal vein system was achieved by injection of 100–150 ml of a barium sulphate suspension (0.6 g/ml) (Fig. 1a + b). Films in a grid cassette were placed directly under the cadaver and exposed with 80 kV/80 mAs (0.6 mm² focus, FFD 90 cm). In addition to films in anterior-posterior projection, angulated views were taken for evaluation of the topographic relation between the opacified bile ducts and veins. In one case a cross section of the hepatoduodenal ligament from the region of the porta hepatis was prepared and the vascular and ductal structures identified by cannulation with polyethylene tubes (Fig. 2).

Die topographische Beziehung der Pfortader zu den extrahepatischen Gallengängen. Eine kombinierte portographisch-cholangiographische Studie an 25 Leichen. Der extrahepatische Verlauf der Pfortader und der Gallengänge sowie deren gegenseitige Lagebeziehungen werden durch simultane Portographie und Cholangiographie an 25 Leichen von Erwachsenen analysiert. Die Befunde werden mit der topographischen Anatomie in der Porta hepatis, im Ligamentum hepatoduodenale und in der duodeno-pankreatischen Region korreliert. An Beispielen wird demonstriert, daß der Abstand der Gallengänge zur Pfortader und zur Vena mesenterica superior in engen Grenzen variiert. Die Untersuchungsergebnisse werden unter Berücksichtigung der klinischen Erfahrung diskutiert, daß Karzinome im proximalen Bereich der extrahepatischen Gallengänge häufig auf die Bifurkation und den Hauptstamm der Pfortader übergreifen, während das Konfluens-Segment der Pfortader und die Vena mesenterica superior bevorzugt von Karzinomen des Pankreaskopfes betroffen werden.

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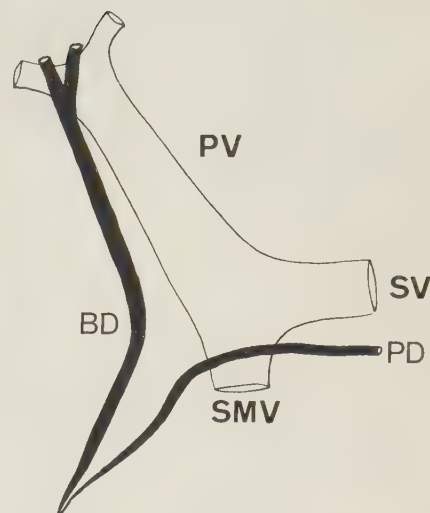


Fig. 1a + b Opacification of splenic vein (SV), superior segment of superior mesenteric vein (SMV), and portal vein (PV) with its intrahepatic ramification. Contrast filling of extrahepatic bile ducts (BD) and pancreatic duct (PD) with side branches. Pancreatic segment of common bile duct passes in region of head of pancreas in gentle deviation to the right before joining pancreatic duct.

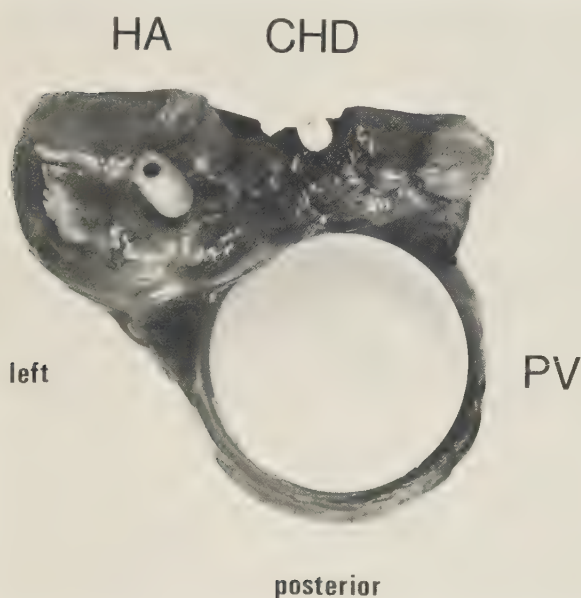


Fig. 2 Cross-section specimen of hepatoduodenal ligament in porta hepatis. Topographic relation between main vascular and ductal structures clearly delineated. Portal vein (PV) posterior to hepatic artery (HA) cannulated with grey catheter, and common hepatic duct (CHD) cannulated with white catheter.

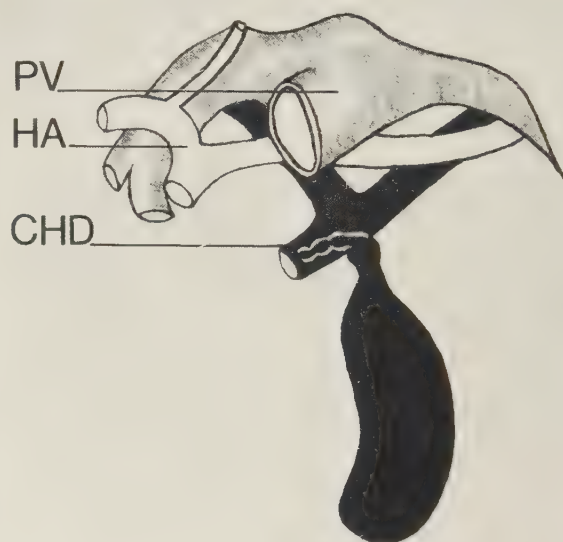


Fig. 3 Posterior aspect of porta hepatis – schematic drawing. Portal vein (PV) with proximal parts of its right and left branches, hepatic artery (HA), and common hepatic duct (CHD).

Anatomical consideration

The following comprehensive survey of the topography of the portal vein and the extrahepatic bile ducts is based on the detailed description given in the anatomical literature (3, 7, 8, 9, 10, 11, 12, 14, 17, 18, 19, 25, 34, 43, 47, 52).

The porta hepatis is situated on the visceral surface of the liver between the quadrate lobe in front and the caudate process behind. Through the porta hepatis the right and left hepatic ducts emerge and the proper hepatic artery and portal vein enter the liver (Fig. 3). The hepatic ducts are situated anteriorly, the portal vein and its right and left branches posteriorly, and the proper hepatic artery with its right and left branches in an intermediate position. The porta hepatis lies enclosed between the two sheets of peritoneum of the free edge of the lesser omentum, which extends

to the liver from the lesser curvature of the stomach (= hepatogastric ligament) and the first part of the duodenum (= hepatoduodenal ligament). The right edge of the lesser omentum lies free between the porta hepatis and the proximal part of the duodenum. It is approximately 3 cm in length and lies in front of the posterior peritoneum, which sweeps over the inferior vena cava. It forms the access to the lesser sac. The right and left hepatic ducts issue from the liver and unite near the right end of the porta hepatis to form the common hepatic duct, which passes downwards for about 3 cm, and is joined in most cases on its right side and at an acute angle by the cystic duct. By the union of the common hepatic duct with the cystic duct the common bile duct is formed. The common hepatic duct is on the right of the proper hepatic artery and in front of the portal vein (Fig. 2). The common



Fig. 4 Schematic drawing of extrahepatic bile ducts showing their topographic relation to portal and superior mesenteric vein and head-neck region of pancreas (modified from P. Belou, 1915) (3).



Fig. 5 Fusion of right and left hepatic ducts (arrow) anterior to bifurcation of portal vein.



Fig. 6 Fusion of right and left hepatic ducts (arrow) anterior to right branch of portal vein.



Fig. 7 Distal part of common hepatic duct and proximal segment (= supraduodenal) of common bile duct in direct contact with portal vein.

bile duct is the direct continuation of the hepatic duct. Its total length varies between 7–9 cm. The first or supraduodenal part passes downward along the right margin of the hepatoduodenal ligament to the right of the hepatic artery, anterior to the portal vein. The second, or retroduodenal part of the duct descends behind the proximal part of the duodenum anterior to the vena cava and to the right of the portal vein. The third, or pancreatic part passes downward, tunneling or grooving the posterior aspect of the head of the pancreas and terminates by piercing the descending duodenum posterior-medially at about its middle. It is separated from the inferior vena cava by connective tissue only or by a thin layer of pancreas. It has no relationship with either the portal vein, which approaches it obliquely from below and from the left, or the superior mesenteric vein, which runs anterior-medially to it at a distance of 2–3 cm (Fig. 4). The posterior-superior pancreaticoduodenal vein (PSPD) issues from the posterior aspect of

the head of the pancreas and ascends upward along the medial margin of the common bile duct to join the portal vein. The portal vein is formed by the junction of the splenic and superior mesenteric veins posterior to the head-and-neck region of the pancreas and anterior to the inferior vena cava. It ascends from behind the duodenum and passes in the free fold of the hepatoduodenal part of the lesser omentum. Its length is approximately 7–8 cm. During its course towards the hilum of the liver it approaches the medial aspect of the extrahepatic bile duct system and in the porta hepatis it is positioned posterior to the junction of the hepatic ducts and the proper hepatic artery.

Results

The anatomic relationship of the biliary ducts and the portal vein is for convenience analysed in three separate divisions: 1. the



Fig. 8 Distal part of common hepatic duct and proximal segment of common bile duct (arrows) separated from portal vein by approximately 1 cm.



Fig. 9 Case demonstrating approximately 2 cm interspace between distal (= pancreatic) segment of common bile duct and superior mesenteric vein. Posterior-superior pancreaticoduodenal vein (PSPD) (arrows) traverses distal segment of common bile duct to join portal vein near its confluence.

porta hepatis, 2. the hepatoduodenal ligament, and 3. the duodenopancreatic region.

1. Porta hepatis – The point of fusion of the right and left hepatic duct was situated anterior to the bifurcation of the portal vein in 7 (Fig. 5) and anterior to the right main branch of the portal vein in 18 (Fig. 6) of 25 cases. In no case was the junction of the hepatic ducts located anteriorly to the left main portal branch. In all cases

the portal vein or its right main branch were in direct contact with the superimposing junction of the hepatic ducts.

2. Hepatoduodenal ligament – Within the approximately 3–4 cm long passage through the lesser omentum, the portal vein kept in close contact with the common bile duct in the majority of cases. Running somewhat medially and posteriorly to the biliary ducts, there was direct contact between these two structures in 7 cases (Fig. 7) and a distance not exceeding 0.5 cm in 14. In 4 cases, however, the common bile duct was separated from the portal vein by an interspace of 1 cm (Fig. 8).

3. Duodeno-pancreatic region – When approaching the duodenum the common bile duct (retroduodenal part) deviated gradually from the portal vein, which continued to descend in an oblique (= inferiomedial) direction, whereas the duct followed a more inferiorly directed course. The distance between the two structures ranged between 0.5–1.5 cm. In the majority of cases the common bile duct descended 1.0 cm to the right of the portal vein, which was in a slightly posterior position in relation to the duct. The region of transition between the retroduodenal and pancreatic sections was in all cases shown to be the most medially located part of the extrahepatic bile duct system. Further on, inferiorly, the common bile duct deviated most often in a gentle curve towards the right before reaching its final (= intramural) segment (Fig. 1). The distance between the pancreatic part of the common bile duct and the distal part of the superior mesenteric vein, i. e. the largest splanchnic vein at this level, ranged between 1.5–3.5 cm (Fig. 9, 10). In 20 of 25 cases this interspace measured between 2.5–3.0 cm. In 9 of 25 cases simultaneous contrast filling of the posteriorsuperior pancreaticoduodenal vein (PSPD) was achieved. Depending on its various points of fusion with the main stem of the portal vein, it followed the right side of the pancreatic and retroduodenal part of the common bile duct 2–3 cm, crossing the bile duct posteriorly before it terminated in the middle part of the portal vein (Fig. 10). In some cases the main stem of the PSPD crossed the pancreatic section of the common bile duct and without accompanying the duct it deviated superior-medially for fusion with the proximal part of the portal vein (Fig. 9). There was no case of ductal or portal venous anomaly in this study.

Discussion

Studying vascular and ductal structures in cadavers implies risk of misinterpretation when transposing the topographic findings without reservation to a living organism. Lack of the physiological intraluminal pressure and tonus of muscular elements within the wall of the vessels and ducts can only insufficiently be compensated for by pressure-controlled postmortal injections. Overfilling may easily result in findings which never exist intra vitam. Aware of these drawbacks, the author undertook to correlate the classical anatomical description of the topography of the extrahepatic bile ducts and their relation to the portal vein and the distal part of its main tributary, i. e. the superior mesenteric vein, with the findings in postmortal portography and cholangiography performed without pressure-recording devices. The findings based on an analysis of x-ray examinations of the region of interest untouched by any disturbing manipulation confirm in general the classical description of the topography of this region. There are, however, some interesting aspects which need to be stressed due to their clinical relevance: Whereas *Belou* (1915) (3) found the hepatic junction anterior to the left main branch of the portal vein in 20 % of cases, we did not see this topographic relation in any of our 25 cases. It is generally accepted that the biliary ducts within the lesser omentum are in “close” relation to the portal vein. In 4 of 25 cases we found a distance of 1 cm between these structures, which may be one of the reasons that a cholangiocarcinoma in this region in single cases does not infiltrate the portal vein. As shown by the venous phase of an arteriographic study of

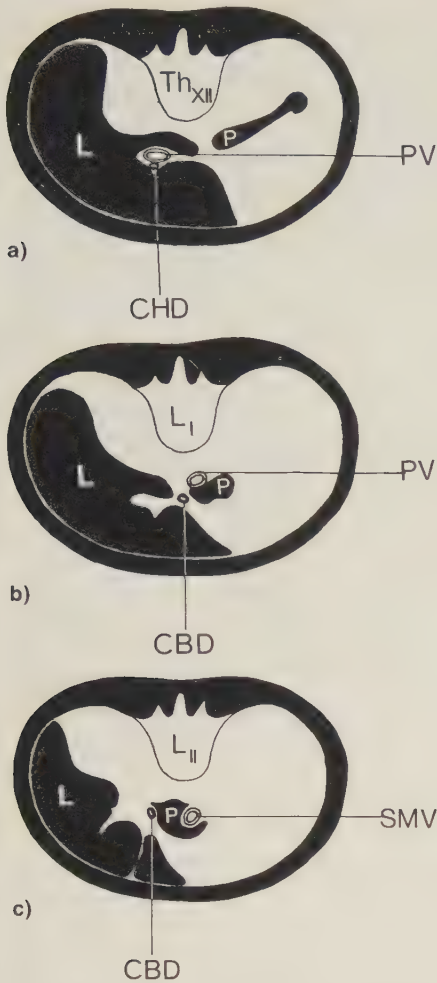


Fig. 11 Cross-sections at levels of ThXII, LI and LII demonstrating topography of extrahepatic bile ducts and portal vein/superior mesenteric vein in coronal plane. The cross-cuts correspond to levels indicated in Fig. 12.
a) Cross-section at level of porta hepatis (A-A in Fig. 12). Portal vein (PV) posterior to and in direct contact with common hepatic duct (CHD). (L) liver. (P) pancreas.
b) Cross-section at level of attachment of hepatoduodenal ligament to duodenum (B-B in Fig. 12). Portal vein slightly posterior-medial to common bile duct (CBD).
c) Cross-section at level of head of pancreas (C-C in Fig. 12). Common bile duct no longer in direct contact with large abdominal veins. Superior mesenteric vein (SMV) anterior-medial to common bile duct.



Fig. 10 Case demonstrating distance of about 3.5 cm between distal segment of common bile duct and superior mesenteric vein. PSPD (arrows) ascends along right margin of pancreatic and retroduodenal segments of common bile duct which it crosses posteriorly before joining middle part of portal vein.

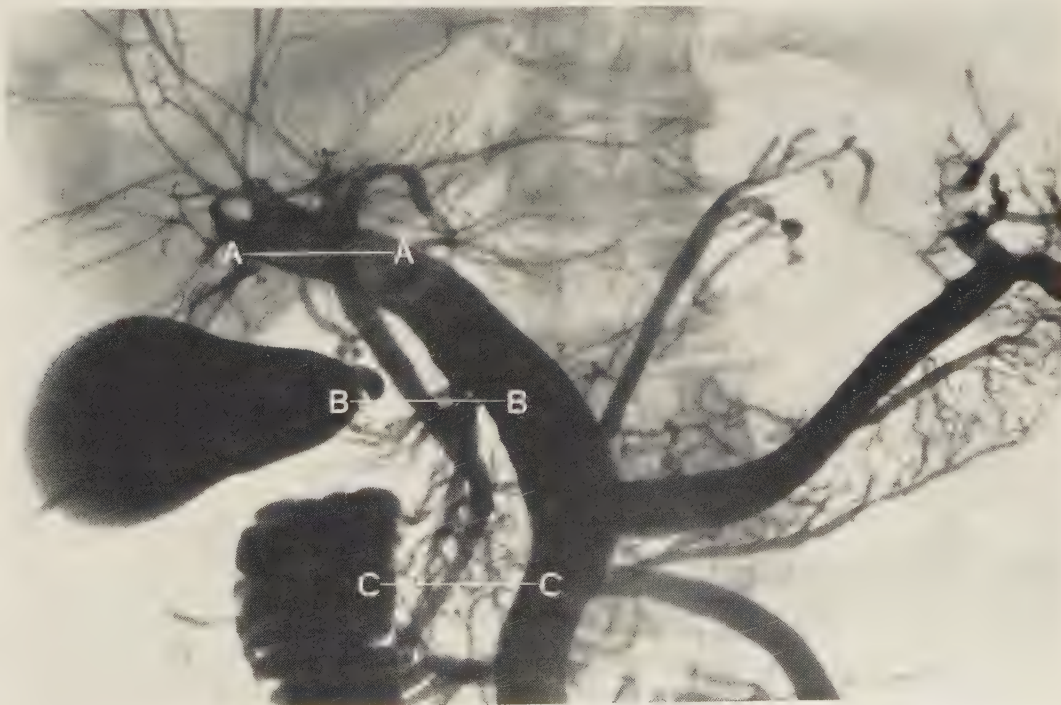


Fig. 12 Simultaneous opacification of gallbladder, biliary duct system, main stem of portal vein, and of splenic, superior, and inferior mesenteric veins. No filling of intrahepatic portal branches due to blood clots. Extensive contrast filling of veins in region of head, body, and tail of pancreas joining portal vein, splenic vein, and mesenteric veins. Cross-sections at levels referred to in Fig. 11.

the superior mesenteric artery or, more distinctly, by percutaneous transhepatic portography (PTP), tumours of the periampullary region may infiltrate or dislodge the confluence of the portal vein and the distal part of the superior mesenteric vein. Whereas in the majority of these cases a carcinoma is present in the head-and-neck region of the pancreas, this encasement of the veins is unusual in a carcinoma originating from the distal segment of the common bile duct or from a papillary tumour. This may be due to the relatively long distance between the papillary/ductal structures and the large splanchnic veins as confirmed by this examination. As far as tumours of the papilla of Vater are concerned, jaundice occurs early when the tumour is still of limited size, explaining the lack of overgrowth to the large abdominal veins. Another aspect of this study may gain clinical interest. In a recent report the possibility of detecting cancer of the head of the pancreas by selective phlebography (44) was demonstrated. This method implies, among others, selective catheterization of the PSPD. As shown by the present investigation, this vein may run in close contact to the distal part of the common bile duct and may be affected by malignant changes originating from its retroduodenal and pancreatic parts. It was demonstrated that the PSPD, confirming the observation of *Falconer* and *Griffiths* (1950) (12), may ascend along the right margin of the distal part of the common bile duct whenever it enters the middle part of the portal vein.

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Percutaneous Transhepatic Portography in Bile Duct Carcinoma. Correlation with Percutaneous Transhepatic Cholangiography and Angiography

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7 Figures

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In 16 patients with carcinoma of the extrahepatic bile ducts, percutaneous transhepatic portography (PTP) and cholangiography (PTC) were performed. Fourteen of these patients also had angiography, which failed to show the tumor in 7 cases. Findings indicating non-extirpability of the tumors were demonstrated by angiography in 3 patients. At PTP non-extirpability was confirmed in these cases. PTP further indicated non-extirpability in one patient and gave hints of infiltration of the liver by the tumor in 4 patients because of intra- or extrahepatic invasion of the portal vein.

When carcinoma is suspected in the extrahepatic bile ducts, a thorough preoperative examination of the biliary duct system and the vessels to the liver may contribute essential information as to the feasibility of radical surgical procedures. As jaundice due to extrahepatic cholestasis is the first symptom in most patients with tumors of the extrahepatic bile ducts (5, 16, 28) cholangiography is indicated for detailed delineation of the intra- and extrahepatic bile ducts. Percutaneous transhepatic cholangiography (PTC) by fine-needle technique has proven to be a highly reliable diagnostic method in jaundice due to extrahepatic cholestasis (1, 22). The proximal margin of a tumor can usually be well defined. A separate puncture of the right and left intrahepatic duct system may be necessary for thorough delineation of tumors of the hepatic confluence. Another important aspect of the percutaneous transhepatic access to the biliary system is the possibility of establishing non-surgical combined internal and external bile drainage (9, 12). By these means the liver may recover from the sequelae of extrahepatic cholestasis prior to the palliative or resective surgical procedures. This is of importance, since there is a direct relation between the preoperative icterus index and postoperative mortality (18). The diagnostic yield of cholangiography is limited, however, to the localization of the tumor within the bile ducts. No information is obtained as to tumor growth outside the ducts and possible encroachment of adjacent arteries and veins.

This retrospective study was carried out to determine if selective examination of the portal vein and its major intrahepatic radicals by percutaneous transhepatic catheterization (PTP) can provide essential information as to the extirpability of bile duct carcinoma beyond the diagnostic findings at angiography.

Material

The series comprised 16 patients: 10 men and 6 women, ranging from 32 to 76 years of age. PTC, angiography and PTP were performed because of jaundice and suspicion of a malignant tumor in the extrahepatic bile ducts. PTC and PTP were

Perkutane transhepatische Portographie bei Gallengangskarzinom. Korrelation mit perkutaner transhepatischer Cholangiographie und Angiographie

Sechzehn Patienten mit Gallengangskarzinom wurden mit perkutaner transhepatischer Portographie (PTP) und Cholangiographie (PTC) untersucht. In 14 Fällen wurde zusätzlich eine Angiographie durchgeführt, die in 7 Fällen keinen Hinweis auf das Vorliegen eines Tumors gab. In 3 Fällen wurde durch Angiographie und PTP nachgewiesen, daß eine radikale Entfernung des Tumors nicht möglich war. Die PTP ergab zusätzlich Hinweise auf einen nicht extirpablen Tumor bei einem Patienten und auf Tumordinfiltration der Leber bei 4 Patienten aufgrund von extra- bzw. intrahepatischer Infiltration der Pfortader.

performed in 16 patients, whereas hepatic and superior mesenteric angiography were carried out in 14 patients. All but one patient were operated on. The mean lapse of time between PTC, which was the first of the invasive diagnostic methods performed in 14 of 16 patients, and operation was 12 days (range 5–21 days). The patient not operated on had advanced tumor spread which was verified at autopsy. In all patients the diagnosis cholangiocarcinoma was verified histologically by operative biopsies (15 patients) or at autopsy (1 patient). Three patients died 1, 2, and 15 days, respectively, following surgery. Autopsy was performed in all 3 cases.

Methods

The percutaneous transhepatic studies (PTC and PTP) and the angiographic examinations were performed after premedication with 10 mg diazepam and 0.5 mg atropine intramuscularly.

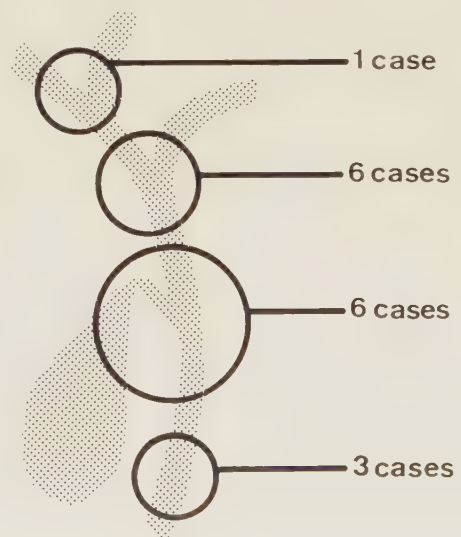


Fig. 1

Site of bile duct occlusion in 16 patients with cholangiocarcinoma.



Fig. 2a PTC: Dilatation of bile ducts of right liver lobe. Due to occlusion proximal to right hepatic duct, contrast filling of various bile duct radicals by separate fine-needle puncture necessary.



Fig. 2b Tumor obstruction passed by catheter with tip in duodenum. Multiple side holes proximal and distal to obstruction warrant internal decompression and drainage of bile duct system of one segment of right liver lobe. Tumor stricture of left hepatic duct close to hepatic confluence.

Fig. 2a-d 39-year-old male (case 1) with cholangiocarcinoma at confluence of hepatic ducts. Infiltration of liver in porta hepatis.

The technique of percutaneous transhepatic intubation and drainage of the bile ducts (9, 12) and of percutaneous transhepatic puncture and catheterization of the portal vein (10, 13) were previously described in detail. Only a brief summary is given here.

For PTC, a fine needle (OD 0.9 mm) was inserted from the right mid-axillary line. If there was dilatation of the bile ducts, a sufficient volume of contrast medium was injected to demonstrate the site of the obstruction. Immediately after the diagnostic procedure, the dilated duct system was punctured from the right mid-axillary line with a 25 cm long stylet fitted with a radiopaque polyethylene catheter (ID OD 1.0/1.6 mm). Using biplane fluoroscopy a peripheral bile duct of the right liver lobe was punctured and a 0.9 mm guide wire with a slightly curved tip was introduced and manipulated towards and – if possible – through the site of the obstruction. The puncture catheter was thereafter removed and a drainage catheter (ID OD 1.4/2.2 mm) inserted. The catheter was passed through the tumor lesion down into the duodenum whenever possible. Multiple side holes proximal and distal to the duct obstruction facilitated internal bile drainage.

Angiography was performed before PTP in all but 2 cases because demonstration of portal vein patency is in general a prerequisite for PTP. The mean lapse of time between angiography and PTP was 10 days (range 0–15 days). In 2 cases, however, the puncture needle unintentionally entered the por-

tal venous system at PTC, and PTP was carried out without angiography.

The portal venous system was also punctured from the right mid-axillary line. For evaluating the splenic, superior mesenteric and portal vein including its major intrahepatic branches, two series of films were taken with the tip of the catheter in the proximal segment of the splenic vein and in the superior mesenteric vein, respectively. Exposures were made with the patient in supine position (40 ml contrast medium, 370 mg I/ml, flow rate 8 ml/s, 5 films/5 s, 8 films/4 s, 3 films/6 s). In addition, a series with the patient in the right and left posterior oblique positions was taken in 4 and 3 cases, respectively. Lateral views with horizontal beam were taken in 4 cases.

The angiographic studies were made by the transfemoral route in all 14 patients. The celiac and superior mesenteric arteries were examined simultaneously with the patient supine in 14 cases (40 ml contrast medium, 370 mg I/ml, flow rate 10 ml/s, 2 films/2 s, 10 films/5 s, 4 films/8 s). For better visualization of the superior mesenteric and portal veins an additional series was taken (60 ml contrast medium, 370 mg I/ml, flow rate 12 ml/s, 8 films/8 s, 8 films/16 s) with the patient in the right posterior oblique position following the injection of 25 mg tolazoline hydrochloride diluted in 10 ml saline in the superior mesenteric artery. The hepatic artery was examined selectively in 10 patients (30 ml contrast medium, 370 mg I/ml, flow rate 8 ml/s, 2 films/2 s, 10 films/5 s, 4 films/8 s) including infusion



Fig. 2c For decompression and drainage, separate catheter placed in dilated bile duct of left liver lobe with ventral approach. Tip of catheter in distal segment of common bile duct. Side holes proximal and distal to tumor stricture of left hepatic duct facilitate internal drainage.



Fig. 2d PTP with simultaneous contrast filling of extrahepatic bile ducts via drainage catheter: Infiltration of main branch of portal vein to right liver lobe about 3 cm distal to its origin (→).

hepatic angiography (50–60 ml contrast medium, 370 mg I/ml, flow rate 2–3 ml/s, 16 films/32 s). The gastroduodenal artery was catheterized in 4 patients (15–20 ml contrast medium, 370 mg I/ml, flow rate approximately 4–6 ml/s, 2 films/2 s, 10 films/5 s, 4 films/8 s). No serious complication was observed following either PTC, PTP or angiography.

The results of the radiographic investigations were related to the operative findings in 15 patients and to the results of post-mortem examinations in 3 of the patients who died postoperatively. In one patient who was not operated on, the radiographic findings were related to those at autopsy (Table I). The approximate tumor size was estimated according to the findings at operation and autopsy. The results of angiography and PTP were analyzed and compared as to their validity in indicating tumor extirpability.

Results

Operative and autopsy findings showed the tumor to be localized in the right hepatic duct in one patient (case 3), at the confluence of the hepatic ducts in 6 patients (cases 1, 2, 4, 5, 6, 7), at the junction of the cystic and common hepatic ducts in 2 patients (cases 8, 9), in the retroduodenal segment of the common bile duct in one patient (case 16), and in the pancreatic segment of the common bile duct in 2 patients (cases 14, 15). In 3 patients (cases 11, 12, 13) the tumor appeared to involve the gallbladder and the proximal segments of the extrahepatic bile ducts, whereas in one patient (case 10) it appeared to affect the gallbladder and the distal segments of the extrahepatic bile ducts. In the latter 4 patients (cases 10, 11, 12, 13) as a consequence of extensive tumor growth it could not be definitely determined if the mass originated primarily from the gallbladder or the extrahepatic bile ducts

(Fig. 1). The detailed findings at operation (15 cases) and/or autopsy (4 cases) are shown in Table I. All of the tumors were adenocarcinomas.

At PTC the site of tumor occlusion was localized in all cases. However, the actual extent of tumor engagement of the intrahepatic ducts was underestimated in all patients with tumors at the confluence of the hepatic ducts (6 cases) and the right hepatic duct (one case). PTC did not demonstrate in 4 cases the total bile duct system of the right liver lobe. The conclusion that this was due to tumor invasion of intrahepatic bile duct radicals was confirmed at operation. The bile ducts of the left liver lobe were not sufficiently filled with contrast medium in 5 of 7 cases when the tumor was localized proximal to or at the confluence of the hepatic ducts. A separate puncture of the left biliary duct system was performed in 2 patients, giving additional information (Fig. 2). In all patients with tumors located distal to the hepatic confluence (9 cases), the ducts in both liver lobes were adequately filled with contrast medium.

The arteriographic examinations were non-conclusive even in retrospect as to the existence of a tumor in 7 patients (cases 1, 3, 4, 6, 13, 15, 16). In 6 of these patients the celiac and superior mesenteric arteries were catheterized whereas in one patient, in addition, a study of the common hepatic artery was made. The tumor size in these patients ranged between approximately 2×2 cm (cases 3, 6, 15, 16) and 4×5 cm (cases 4, 13). Single tiny vessels strongly suggesting neoplastic origin or tumor-infiltrated arteries, were demonstrated in 4 patients close to the bile duct stricture shown at PTC (cases 2, 8, 14) or in the area of the gallbladder (case 11). Tumor encasement of the hepatic arteries close to the hilum of the liver was found in 2 patients (cases 10, 12; Fig. 3). The gastroduodenal artery

Table I Summary of findings at PTC, PTP, angiography and operation/autopsy in 16 patients with carcinoma of the extrahepatic bile ducts

Case No.	Age/ Sex	Site and extent of tumor based on findings at surgery and/or autopsy	PTC	PTP	Angiography	Surgical procedure	Comment
1	39M	2 × 3 cm tumor in porta hepatis. Infiltration of liver in porta hepatis	Occlusion of both hepatic ducts at their confluence	Portal vein branch to right liver lobe infiltrated appr. 3 cm distal to its origin	No pathologic findings	Extended right hemihepatectomy. Remnant of left hepatic duct anastomosed with common bile duct	Tumor not radically removed
2	66F	3.5 × 3.5 cm tumor in porta hepatis. Marked infiltration of right hepatic duct	Occlusion of both hepatic ducts at their confluence	No pathologic findings	Single abnormal vessels close to bile duct stricture	Right hemihepatectomy. Partially resected left hepatic duct anastomosed with common bile duct	Tumor not radically removed
3	55M	2 × 2 cm tumor in porta hepatis. Infiltration of portal vein branch to right liver lobe	Occlusion of right hepatic duct close to confluence. Duct system of left lobe not demonstrated	Portal vein branch to ventrocranial segment infiltrated	No pathologic findings	Right hemihepatectomy. Left hepatic duct anastomosed with common bile duct	Pat. 4 years later alive. No symptoms of tumor recurrency
4	76M	4 × 5 cm tumor in porta hepatis	Occlusion of right hepatic duct. Duct system of left lobe not demonstrated	Extremely fine but regular portal veins in small left liver lobe	No pathologic findings	Exploratory laparotomy. Tumor not extirpable	Autopsy 1 day after operation, left liver lobe, appr. 4 × 5 × 3 cm with dilated bile ducts
5	32M	5 × 8 cm tumor in porta hepatis. Infiltration of left liver lobe. Infiltration of portal vein branch to left liver lobe	Occlusion of right hepatic duct close to confluence. Duct system of left lobe not demonstrated	Occlusion of left branch of portal vein at its origin	Multiple abnormal vessels in appr. 5 × 5 cm area in and cranial to porta hepatis. Portal vein compressed in liver hilum. Occlusion of portal vein branch to left liver lobe suspected	Left hemihepatectomy. Resection of central bile ducts. Bile drainage of right liver lobe via percutaneous catheter	Pat. expired 3 weeks after operation. No autopsy performed
6	37F	2 × 3 cm tumor in porta hepatis	Occlusion of both hepatic ducts at their confluence	No pathologic findings	No pathologic findings	Resection of hepatic confluence. Remnant of left hepatic duct anast. with common bile duct. Right hepatic duct closed. Biliary ducts of right liver lobe drained via percutaneous trans-hepatic catheter	Autopsy 7 months after operation: 6 × 7 cm tumor in porta hepatis. Infiltration and obstruction of portal vein
7	75M	2.5 × 3 cm tumor in porta hepatis. Tumor growth predominantly in left hepatic duct. Infiltration of portal vein branch to left liver lobe	Occlusion of both hepatic ducts at their confluence. Duct system of left lobe not demonstrated	Infiltration of left branch of portal vein close to its origin	Not performed	Exploratory laparotomy. No tumor resection performed. Insertion of transhepatic drainage tube in common bile duct	Autopsy 4 days after operation: tumor limited to porta hepatis
		Large tumor in porta hepatis. Tumor growth adjacent to portal vein	Occlusion of common hepatic duct	Compression of portal vein branch to dorsocaudal segment of right liver lobe	Encasement of single minor arteries in right liver lobe close to porta hepatis	Exploratory laparotomy. No tumor resection performed. Bile drainage via tubes in right and left hepatic ducts	

Table I Summary of findings at PTC, PTP, angiography and operation/autopsy in 16 patients with carcinoma of the extrahepatic bile ducts

Case No.	Age/ Sex	Site and extent of tumor based on findings at surgery and/or autopsy	PTC	PTP	Angiography	Surgical procedure	Comment
9	76F	5 × 6 cm tumor engaging cystic and common hepatic ducts. Infiltration of liver in porta hepatis	Occlusion of common hepatic duct	Occlusion of portal vein branch to dorsocaudal segment of right liver lobe	Not performed	Exploratory laparotomy. No tumor resection performed. Transhepatic tube drainage of bile ducts	Autopsy 2 days after operation: Common bile duct and gallbladder free of tumor growth
10	76F	Large tumor engaging gallbladder, extrahepatic bile ducts, duodenum and head of pancreas	Occlusion of common bile duct	Marked infiltration of main stem of portal vein	Encasement of left hepatic artery at its origin. Encasement of gastroduodenal artery and pancreatic arcades. Main stem of portal vein compressed	No operation performed	Autopsy: tumor growth along common bile duct with infiltration of portal vein
11	44M	4 × 6 cm tumor engaging gallbladder, common hepatic duct and hepatic confluence	Stricture of common bile duct	No pathologic findings	Encasement of cystic arteries	Exploratory laparotomy: No tumor resection performed. Operative insertion of bile duct endoprosthesis	—
12	46F	6 × 7 cm tumor engaging gallbladder. Infiltration of hepatoduodenal lig. Occlusion of common hepatic duct	Occlusion of common hepatic duct	Marked stenosis of main stem of portal vein close to liver hilum	Encasement of left and middle hepatic arteries at their origin. Tiny abnormal vessels in area between liver hilum and head of pancreas. Stenosis of main stem of portal vein close to liver hilum	Cholecystectomy. No tumor resection performed. Transhepatic tube drainage of bile ducts	—
13	67F	4 × 5 cm tumor engaging gallbladder, cystic duct, common hepatic and common bile ducts	Marked stricture of supra- and retroduodenal segments of common bile ducts	No pathologic findings	No pathologic findings	Exploratory laparotomy. No tumor resection performed. Bile drainage via tube through tumor stricture till duodenum	—
14	63M	2.5 × 2 cm tumor at distal (= intrapancreatic) segment of common bile duct. Tumor growth adjacent to portal vein	Occlusion of distal segment of common bile duct	Partial compression of proximal segment of portal vein	Abnormal vessels close to stricture of common bile duct. Encasement of gastroduodenal artery	Pancreatectomy. Duodenectomy. Partial resection of common bile duct, choledochojejunostomy	Tumor not radically removed
15	67M	2 × 3 cm tumor at distal (= intrapancreatic) segment of common bile duct. Tumor growth into wall of portal vein	Occlusion of distal segment of common bile duct	No pathologic findings	No pathologic findings	Pancreatectomy. Partial resection of common bile duct, choledochojejunostomy	Tumor not radically removed. Infiltration of portal vein not shown at PTP
16	69M	2 × 2 cm tumor at retroduodenal segment of common bile duct	Occlusion of distal segment of common bile duct	No pathologic findings	No pathologic findings	Partial resection of common bile duct, choledochojejunostomy	Tumor not radically resected

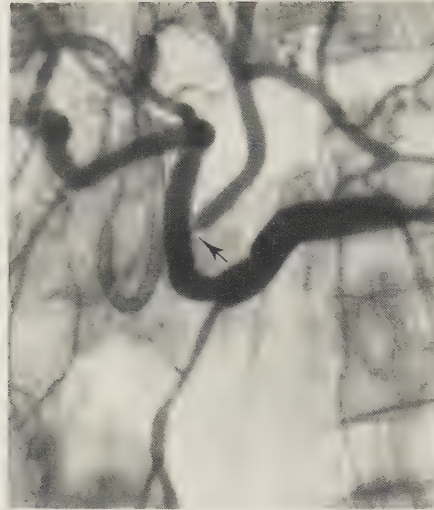


Fig. 3 a

Fig. 3 b

Fig. 3 c

Fig. 3a–c 75-year-old female (case 10) with cholangiocarcinoma engaging gallbladder, extrahepatic bile ducts, and head of pancreas.

Fig. 3a PTC: Occlusion of common bile duct with dilatation of intrahepatic and proximal segments of extrahepatic bile ducts.

Fig. 3b Angiography of common hepatic artery: Encasement of left hepatic artery at its origin (→). Encasement of proximal part of gastroduodenal and cystic arteries. Percutaneous transhepatic drainage catheter proximal to bile duct occlusion.

Fig. 3c PTP – anterior-posterior projection: Marked stenosis of portal vein at its midpoint due to tumor infiltration.

was encased by the tumor in 2 patients. Multiple neoplastic vessels were observed intrahepatically close to the porta hepatis in one patient (case 5; Fig. 4). The venous phase of the simultaneous study of the celiac and superior mesenteric arteries following injection of tolazoline hydrochloride into the superior mesenteric artery resulted in all 14 cases in visualization of the splenic vein, the main stem of the superior mesenteric vein, and the portal vein. The technical quality of the indirect (= arterial) portogram ranged widely. No conclusion as to detailed morphology was possible in 6 cases because of low concentration of the contrast medium. However, no marked gross pathological changes in location and diameter of the portal vein or possible compression/infiltration of its main branches were observed. No tumor changes were observed in 5 cases with good delineation of the portal vein and the proximal segments of its intrahepatic branches. Partial compression of the portal vein in the liver hilum with possible occlusion of the main branch to the left liver lobe at its origin was demonstrated in one patient (case 5). There was marked compression of the main stem of the portal vein in two patients (cases 10, 12). A summary of the findings is given in Table I.

At PTP the confluence of the portal vein, its main stem, and the proximal segment of its intrahepatic branches were distinctly demonstrated in all 16 patients. No pathologic findings were shown in 7 patients (cases 2, 4, 6, 11, 13, 15, 16). In 4 of these patients the tumor was located in the confluence of the hepatic ducts (cases 2, 4, 6, 11), in one patient (case 13) the common hepatic, cystic and common bile ducts and in 2 patients (cases 15, 16) the pancreatic and retroduodenal segments of the common bile duct were engaged by the tumor. Partial compression, infiltration or occlusion of the main portal vein branch to the right and left lobe was shown in one (case 1) and 2 patients, (cases 5, 7) respectively, in whom the tumor was located at the confluence of the hepatic ducts (Figs. 2, 4). Compression and infiltration of the portal vein branch to the

ventrocranial and dorsocaudal segments of the right lobe were present in one and 2 patients, respectively (cases 3, 8, 9). The tumors were located close to the confluence of the hepatic ducts. Compression and infiltration of the main stem of the portal vein was demonstrated in 3 patients (cases 10, 12, 14) in whom the tumor was located distal to the confluence of the hepatic ducts (Figs. 3, 5).

Findings at angiography and PTP related to findings at surgery

Findings indicative of non-extirpability of the tumor were found in 3 (cases 5, 10, 12) of 14 patients in whom angiography was performed. In 2 of these (Figs. 3, 5) tumor encasement of the hepatic arteries was present in the area of the porta hepatis. In one of these patients (case 10) the tumor in addition encased the gastroduodenal artery. In one patient (Fig. 4) a large, mainly intrahepatic tumor was demonstrated cranial to the liver hilum. In these 3 patients tumor infiltration and partial compression of the portal vein or its branches was demonstrated at indirect (= arterial) portography and confirmed at PTP. Two of these patients were operated on. A left hemihepatectomy was performed in one patient (case 5) although the infiltrating tumor growth excluded curative surgery. The right liver lobe was drained through a percutaneous transhepatic catheter. The other patient (case 12) received as palliation a transhepatic tube drainage of the biliary system. The tumor was deemed non-resectable at operation. The third patient (case 10) died prior to surgery. A large tumor was found at autopsy involving the gallbladder, the structures in the lesser omentum and the duodenum.

In 4 patients (cases 2, 8, 11, 14) in whom arteriography showed fine neoplastic vessels and/or tumor-encased arteries, PTP was normal in 2 patients (cases 2, 11) whereas portal vein infiltration was present in the remaining 2 patients (cases 8, 14; Fig. 7). Extensive surgery was performed in 2 of these patients (cases 2, 14). Microscopy disclosed in both cases that the tumor was not totally removed. In the other 2 patients

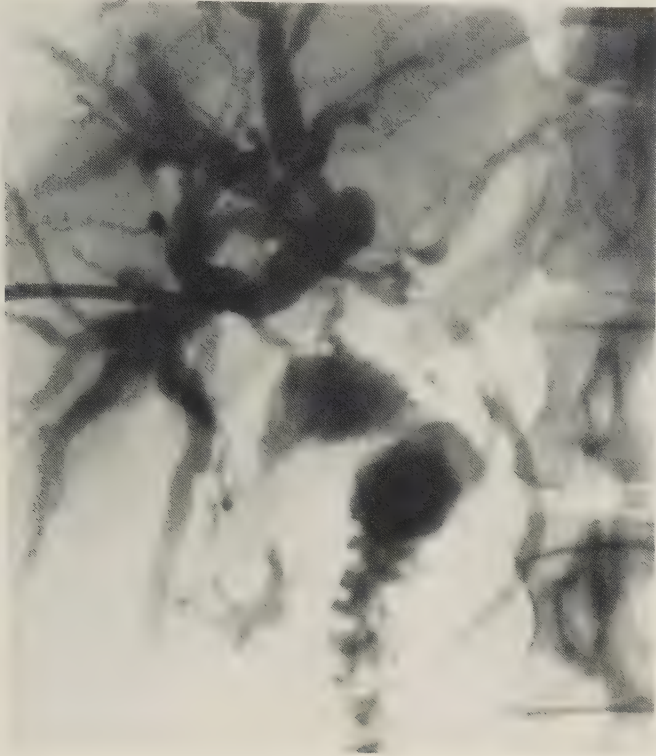


Fig. 4a PTC: Stricture of biliary system in porta hepatis. Dilatation of bile ducts of right liver lobe. No contrast filling of bile ducts of left liver lobe. Common bile duct normal.



Fig. 4b Angiography of celiac artery: Multiple abnormal vessels in tumor area (→) in and cranial to porta hepatis.

Fig. 4a–c 32-year-old male (case 5) with approximately 5 × 8 cm cholangiocarcinoma obstructing confluence of hepatic ducts.

(cases 8, 11) a bile duct endoprosthesis was operatively inserted as a palliative measure because of non-resectability of the tumor.

In the remaining 7 patients angiography provided no information as to the existence of a tumor (cases 1, 3, 4, 6, 13, 15, 16). One of these patients (case 3) is alive and without symptoms of tumor recurrence 4 years after right hemihepatectomy and construction of an anastomosis between the left hepatic and common bile ducts. In 4 patients (cases 1, 6, 15, 16: Figs 2, 6) tumor resection was performed. According to the microscopic examination of the operation specimens, however, the tumor was not radically removed in any of these cases.

In 2 patients (cases 1, 3: Fig. 2) where angiography was normal, PTP showed intrahepatic tumor engagement of the portal vein. Extensive surgical procedures were performed in both cases. One patient (case 3) is free of tumor recurrence 4 years after operation, whereas the tumor could not be removed radically in the other patient (case 1: Fig. 2). In 5 patients (cases 4, 6, 13, 15, 16: Fig. 6) both angiography and PTP were normal. In 3 of these (cases 4, 6, 13) the tumor was graded as non-extirpable at operation, whereas extensive surgery was performed in the other 2 patients. In both cases the tumor tissue was not radically removed, however.

In 2 patients (cases 7, 9) in whom no angiography was performed PTP showed infiltration and occlusion of the left main branch of the portal vein (case 7) and occlusion of the dorsocaudal segmental branch of the right lobe (case 9). At operation the tumor was regarded as non-extirpable in both cases. A summary of the findings at PTP/angiography with correlation to tumor extirpability is given in Table II.



Fig. 4c PTP: Occlusion of left branch of portal vein at its origin because of tumor infiltration. Partial compression of right branch of portal vein.

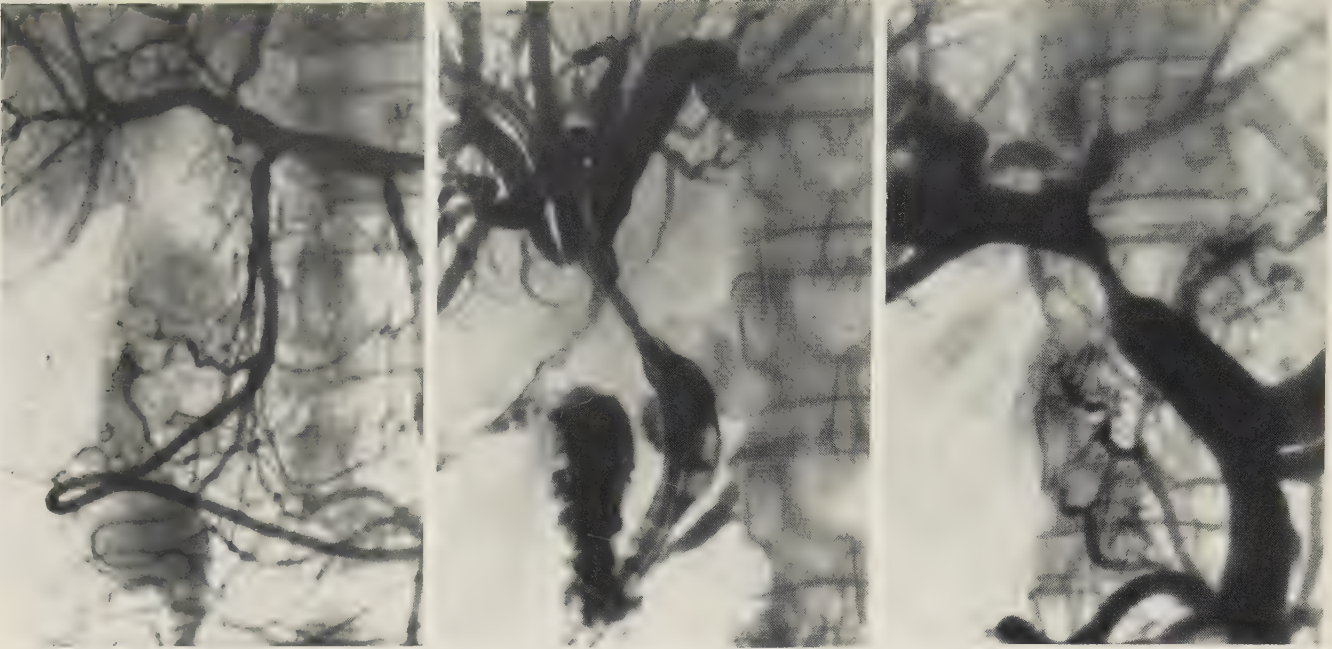


Fig. 5 a Angiography of common hepatic artery: Encasement of left and middle hepatic arteries at their origin. Multiple tiny abnormal vessels in area between liver hilum and head of pancreas. Percutaneous transhepatic drainage catheter proximal to bile duct occlusion.

Fig. 5 b Cholangiography via operatively placed transhepatic drainage tube: Stricture of common hepatic duct. Dilatation of intrahepatic bile ducts. Stones in moderately dilated common bile duct. Distal segment of pancreatic duct filled with contrast medium.

Fig. 5 c PTP – anterior-posterior projection: Marked stenosis of main stem of portal vein close to liver hilum due to tumor infiltration.

Discussion

A common finding in carcinoma of the extrahepatic bile ducts is local invasion by the primary lesion rather than distant metastases. The close proximity of the extrahepatic bile ducts to the hepatic artery(ies) and the portal vein in the hilum of the liver may result in early involvement of these vital structures, which excludes tumor extirpability (i.e. curative surgery). In a topographic study (11) the distance between the bile ducts and the portal vein in the hepatoduodenal ligament was found to range between 0–1 cm. In the duodeno-pancreatic region the interspace between the common bile duct and the portal and superior mesenteric veins ranged between 0.5–3.5 cm. When reviewing clinical reports on the question of surgery in patients with carcinoma of the extrahepatic bile ducts, non-

extirpability of tumors due to infiltration of the portal vein is frequently found (3, 4, 6, 14, 17, 23, 30, 31, 36). Portal vein obstruction with sequelae of portal hypertension as a rare and usually late manifestation of cholangiocarcinoma is on record (7). An analysis of the frequency of portal vein involvement by carcinoma of the extrahepatic bile ducts does not seem to exist. This may be explained by the fact that even extensive dissection in many cases cannot rule out tumor engagement of the vascular structures. Ross et al. (25) and Thorbjarnarson (30) reported a massive fibroblastic reaction stimulated by the carcinoma of the proximal bile ducts in the surrounding tissues. Therefore tumor involvement of the hepatic artery and portal vein cannot be excluded unless microscopy is performed.

Table II Correlation of findings at PTP/angiography with tumor resectability

	Angiography	Encasement of hepatic and/or gastroduodenal artery	Tumor vessel and/or small artery encasement	Normal findings	Total
PTP					
Tumor infiltration of portal veins		3 (3)	2 (2)	2 (1)	7 (6)
Normal findings		0	2 (2)	5 (5)	7 (7)
Total		3	4 (4)	7 (6)	14 (13)

Extirpable or incompletely removed tumors. In two additional patients only PTP was performed. Invasion of portal vein branches was observed in both cases. At operation the tumors were found to be non-extirpable



Fig. 6a PTC – left anterior oblique projection: Distal segment of common bile duct occluded. Marked dilatation of intrahepatic and proximal segments of extrahepatic bile ducts. Percutaneous transhepatic drainage catheter proximal to bile duct occlusion.



Fig. 6b Simultaneous angiography of celiac and superior mesenteric arteries: Main stem of splanchnic arteries dislodged to left due to previous partial resection of stomach. No findings indicating tumor.

Fig. 6a–c 67-year-old male (case 15) with approximately 2 × 3 cm cholangiocarcinoma originating from distal (= pancreatic) segment of common bile duct.

Various authors (8, 15, 24, 35) have investigated the diagnostic effectiveness of angiography in cholangiocarcinoma of the extrahepatic bile ducts. The present study did not confirm the high diagnostic yield reported by others (15, 24). Whereas angiography can provide essential information in determining the probable nature and resectability of a lesion, PTC is superior in defining the exact site of obstruction and the anatomy of bile ducts (2). However, it is evident from this study that even tumors beyond the limits of curative surgery may escape angiographic detection. Several authors (2, 8, 15, 24, 35) have pointed out that invasion of large veins, particularly the portal vein, is an uncommon finding at angiography of carcinoma of the extrahepatic bile ducts. Röscher (26), however, observed occlusion of the portal vein in 4 of 9 patients with carcinoma of the extrahepatic bile ducts and the gallbladder. The results of the present study suggest that the tumor growth into the wall of the portal vein is underestimated at angiographic examinations. Even phar-maco-enhanced arterial (= indirect) portography may result in insufficient demonstration of the detailed morphology of the portal vein as shown in this investigation. In addition, considering the normal topographic relation between the bile ducts and the portal vein within the porta hepatis with the ducts lying directly anterior to the portal vein, it is evident that early encroachment of the portal vein may be detected only when the examination is carried out in the lateral projection. In 4 patients reported herein PTP was performed with horizontal beam in supine position. One of these patients had tumor invasion of an intrahepatic portal vein branch. This was better demonstrated in anterior-posterior projection, however. Finally, there is evidence that even PTP, which is superior to arterial (= indirect) portography because of the higher concentration of contrast medium in the portal vein, may fail to demonstrate existing tumor growth into the wall of the portal vein as shown in one patient (Fig. 6). Tumor affection of the portal venous system was demonstrated by PTP in 9 of the 16 patients reported herein. In 7 of these patients arteriogra-



Fig. 6c PTP: Confluence of portal vein displaced to left due to previous partial resection of stomach. No signs of tumor infiltration. At operation, tumor adherent to proximal segment of portal vein.

phy was performed and revealed tumor infiltration/compression of the portal vein in 3 cases only. Frequently a carcinoma originating from the extrahepatic ducts in the porta hepatis extends deep along a hepatic duct and into the parenchyma of the liver (32). Intrahepatic portal vein branches may be infiltrated or compressed by the expanding tumor. In the material presented herein, PTP, but not angiography, disclosed infiltration or compression of portal vein branches to the liver lobes or their segments in 4 patients (cases 1, 3, 8, 9; Fig. 2). Angiography and PTP should be performed when bile drainage has been well established. Dislo-



Fig. 7a PTC: Bile duct occluded distal to origin of cystic duct. Marked dilatation of intrahepatic and proximal segments of extrahepatic bile ducts. Percutaneous transhepatic drainage catheter proximal to bile duct occlusion.

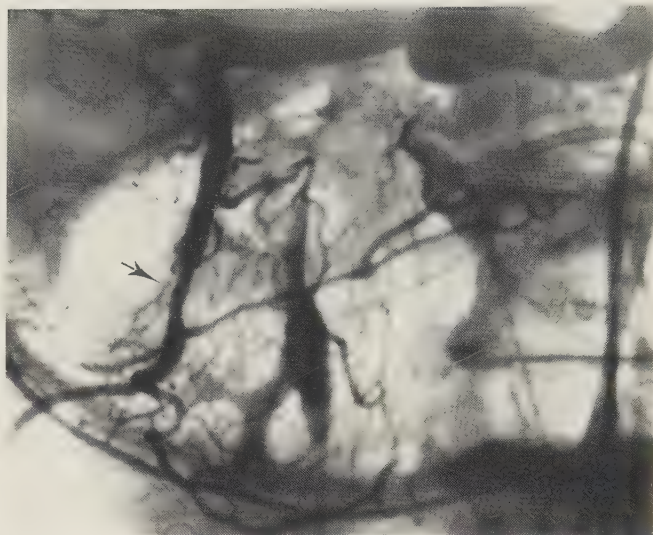


Fig. 7b Angiography of celiac artery – left anterior oblique projection: Abnormal vessels close to slightly encased gastroduodenal artery (→).

Fig. 7a–c 63-year-old male (case (14) with approximately 2.5×3 cm cholangiocarcinoma originating from distal (= pancreatic) segment of common bile duct



Fig. 7c PTP: Slight compression of proximal segment (→) of portal vein. At operation tumor adherent to portal vein in this area.

cation and compression of vascular structures due to dilated bile ducts may otherwise be considered a sign of tumor displacement and/or infiltration. A hematoma may develop in the hepatoduodenal ligament following PTC (34) and be another cause of diagnostic error when compressing or displacing the portal vein. It may be difficult to decide if there is true infiltration of the portal venous system by a tumor when a smooth compression and/or a dislocation of the vein is observed at PTP or angiography. However, there is strong evidence that even the slightest sign of venous compression (Fig. 7) may be equivalent to infiltration of the respective vein. In 6 patients (cases 1, 3, 5, 7, 8, 10) mild-to-marked compression of different segments of the portal venous system at PTP was found to represent tumor infiltration of the vessels at

surgery/autopsy and/or microscopy (Figs. 2, 3, 4). *Sato et al.* (27) examined 11 patients with intrapancreatic bile duct carcinoma preoperatively with splenoportography and found compression of the portal vein in 3. At operation non-extirpable tumors were found and only palliative resections could be performed. *Marions et al.* (19, 20) concluded on the basis of their experience with direct (transumbilical or percutaneous transhepatic) portography in patients with carcinoma of the bile duct and the pancreas that compression of the portal vein does not imply infiltration by the tumor. However, the only convincing evidence for this statement was not presented, i.e. the microscopic examination of the segment of the portal vein involved by tumor compression.

Norton et al. (21) resected the portal vein in 3 patients. It was surrounded but not penetrated by pancreatic carcinoma. The carcinoma invaded the wall of the portal vein in one case and the adventitia of the portal vein in 2 cases.

These findings give reason to postulate that a malignant tumor adjacent to the portal vein is not radically removed unless en bloc resection of the tumor and the tumor-affected vein is carried out. Tumor infiltration of the portal vein is traditionally regarded as a contra-indication to tumor resection. Nevertheless *Fortner et al.* (6) performed en bloc resection of bile duct carcinoma. This operation included resection of the involved portions of the portal vein, with vascular reconstruction by re-anastomosis of the portal vein (2 cases) or construction of a proximal end-to-side porto-caval shunt (one case).

In carcinoma of the bile ducts at the porta hepatis a right or left hepatic lobectomy is indicated if spread of the carcinoma involves the right or left intrahepatic bile ducts beyond the second bifurcation (14). It was therefore of interest to evaluate the effect of tumor engagement of the intrahepatic portal vein branches on the final outcome of surgery: In 4 patients (cases 1, 3, 8, 9) the distal part of the right main branch of the portal

vein or one of its segmental branches was involved by the tumor lesion as demonstrated at PTP. In one of the patients (case 1) a 2–3 cm tumor in the porta hepatis infiltrated the liver at its hilum. Although the right liver lobe was resected, the tumor was not removed radically. In 2 patients (cases 8, 9) the tumor occluded the common hepatic duct and was deemed non-resectable because of growth adjacent to the portal vein in the liver hilum or infiltration of the liver at its hilum, respectively. In one patient (case 3) the tumor was localized in the porta hepatis occluding the right hepatic duct. A right hepatic lobectomy was performed and the left hepatic duct was anastomosed to the common bile duct. The patient is well and free of symptoms of tumor recurrence 4 years after operation. Consequently tumor infiltration of intrahepatic portal vein branches does not necessarily exclude curative surgery, provided that the main stem of the portal vein is free of tumor growth. However, in all patients with tumor involvement of the extrahepatic portal venous system, demonstrated at angiography and/or PTP, the lesion was not radically extirpable. The criteria of extirpability in carcinoma of the extrahepatic bile ducts do not differ from those applied in carcinoma of the head of the pancreas. Regardless of the surgical method applied, involvement of the mesenteric and hepatic arteries (29, 33) as well as of the portal and mesenteric veins definitely rules out curative tumor surgery. It should be clear, that none of the tumors without invasion of the portal vein or its branches were completely removed at operation. On the other hand, if there is tumor infiltration of intrahepatic portal vein branches liver resection may be curative (case 3).

Conclusion

Angiography indicated non-extirpability of the tumor lesion in 3 of 14 patients because of encasement of major vessels. This information was obtained from the arterial phase alone in one patient and from the arterial and venous phases in 2. Non-extirpability was confirmed at PTP in these cases. In addition, PTP indicated non-extirpability in one patient and gave hints of infiltration of the liver by the tumor in 4 patients because of intra- or extrahepatic invasion of the portal vein. This information was not gained by arteriography. At operation tumors were regarded as non-extirpable in 7 patients where neither arteriography nor PTP gave this information. PTP as a complement to arteriography may provide information as to tumor extirpability when tumor infiltration of the portal venous system is demonstrated. Negative findings at PTP, however, do not exclude tumor growth along and in the wall of the portal vein and are therefore of little importance in the preoperative evaluation of tumor extirpability.

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PERCUTANEOUS TRANSHEPATIC INSERTION OF A
PERMANENT ENDOPROSTHESIS IN OBSTRUCTIVE
LESIONS OF THE EXTRAHEPATIC BILE DUCTS

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ABSTRACT

A teflon endoprosthesis for permanent bile drainage was inserted in 13 patients following percutaneous transhepatic puncture and catheterization of the bile duct system. Twelve patients had extrahepatic cholestasis because of a malignant tumor, whereas one patient had chronic inflammation involving the hepatoduodenal ligament (secondary to Crohn's disease) with obstruction of the extrahepatic bile ducts. The drainage periods varied from one week to eight months. The endoprosthesis was regarded as partially effective in seven patients whereas in six cases the drainage through the endoprosthesis was insufficient and external bile drainage through a percutaneous transhepatic catheter was necessary. Infection of the bile duct system during the drainage period with a percutaneous transhepatic catheter and/or bile duct endoprosthesis occurred in ten patients. Spontaneous dislocation of the endoprosthesis occurred in five patients in varying degrees. One patient developed an intrahepatic aneurysm adjacent to the puncture tract and died because of liver insufficiency following therapeutic embolization of the aneurysm and most of the hepatic arteries by injection of gel foam particles into the common hepatic artery. Patients in whom palliative treatment by insertion of a permanent bile duct endoprosthesis may be suitable were defined.

Key words: Malignant extrahepatic cholestasis - Bile duct endoprosthesis, percutaneous transhepatic insertion - Bile duct endoprosthesis, function and complication.

INTRODUCTION

The percutaneous transhepatic approach to the biliary ducts, initially described for diagnostic cholangiography (PTC) (1 - 4), has also had increasing therapeutic application in extrahepatic cholestasis caused by malignant tumors or postoperative stricture. Various techniques for external bile drainage by percutaneous transhepatic catheter intubation of the duct system have been described (5 - 15). Further technical refinement has made it possible to manipulate percutaneous transhepatic catheters through the obstructing lesion into the distal segment of the common bile duct or the duodenum. This facilitates combined external/internal bile drainage (16 - 24). The subsequent creation of a non-surgical biliodigestive communication by percutaneous transhepatic insertion of a bile duct endoprosthesis through the malignant stricture of the extrahepatic bile ducts has also been reported (25 - 28).

Based on experience with temporary and permanent combined internal/external bile drainage via percutaneous transhepatic catheters (18,19), a technique was developed for non-surgical introduction of a permanent bile duct endoprosthesis modified from that used by other authors (25 - 28). The aim of the present investigation was to present a critical assessment of this technique and its clinical applicability.

MATERIAL

The study comprised 13 patients, eight women and five men, ranging from 22 to 90 years of age. All patients presented with jaundice subsequently found to be caused by extrahepatic cholestasis. Obstruction of the extrahepatic bile ducts by a malignant tumor was verified in 12 cases. Fine-needle aspiration biopsy from the site of biliary obstruction was performed in connection with PTC in 10 cases and following angiography in one case. Cytology verified a malignant lesion in all of these patients. In one patient a malignant tumor was verified histologically first at autopsy.

Only one of the patients with a malignant tumor obstructing the extrahepatic bile duct was operated on prior to the insertion of a bile duct endoprosthesis. The construction of a bilio-digestive anastomosis was regarded as not feasible, however. In one patient with Crohn's disease, extrahepatic cholestasis was caused by a benign bile duct stricture. At operation an inflammatory mass was found involving the porta hepatis, the hepatoduodenal ligament, and the duodenum. In addition, there was inflammation of the terminal segment of the ileum. A gastroenterostomy was performed because of duodenal stricture. Exploration of the hepatoduodenal ligament was complicated by a tear lesion of the portal vein. Surgical decompression of the extrahepatic cholestasis by a bilio-digestive anastomosis was deemed too hazardous because of the risk of fistula formation and recurrence of the stricture.

Angiography was performed in four patients. Signs of a malignant tumor were observed in the region of the liver hilum in one patient and in the head of the pancreas in three patients. In all these patients encasement of major arteries was present. In addition, changes suggesting liver metastases were observed by computer tomography and percutaneous transhepatic portography in one patient each. Surgical procedures were regarded as contraindicated in the remaining five elderly patients because of their poor general condition. The platelet count was above 50,000/mm³

and APTT (Activated Partial Thromboplastine Time) below 35 seconds in all patients when the endoprosthesis was placed in position.

METHOD

In all patients in this series, combined internal/external bile drainage was performed via a percutaneous transhepatic catheter prior to the insertion of a bile duct endoprosthesis. The drainage period ranged between eight days and 11 weeks (mean 4 weeks) in 12 patients with malignant tumors. One patient with an inflammatory mass in the hepatoduodenal ligament had bile drainage through a catheter for nine months. The technique of combined internal/external bile drainage via a percutaneous transhepatic catheter was previously described in detail (18,19).

A teflon tube (ID/OD 3.0/4.0 mm, William Cook ApS, Denmark) was used as endoprosthesis. The proximal end of the tube was slightly flared giving an outer diameter varying between 5 and 6 mm. The distal end was slightly tapered. Nine of 13 endoprostheses in this material were furnished with shallow grooves at 1 cm intervals. The length and shape of the endoprosthesis was chosen according to the site and location of the bile duct stricture/occlusion in the individual case (Fig. 1).

Technique of insertion of bile duct endoprosthesis

The procedure required no general anesthesia. The patients were premedicated with 10 mg diazepam and 0.5 mg atropine intramuscularly. Immediately prior to the dilatation of the tumor stricture and the insertion of the endoprosthesis an additional 5 mg diazepam was given intravenously. Passage through the bile duct stricture/occlusion by a guide wire and catheter was the prerequisite for the percutaneous transhepatic insertion of the bile duct endoprosthesis. A specially designed hard guide wire (Surgimed, Denmark) (29) with a 10 cm flexible segment at its tip was introduced into the indwelling drainage catheter until the tip of the guide wire reached the duodenum. Following local anesthesia around the puncture canal in the skin, subcutis and intercostal muscles, the drainage catheter was removed with the guide wire tip remaining in the duodenum. The bile duct stricture/occlusion was widened by introduction of two coaxial dilatation teflon catheters (William Cook ApS, Denmark). Catheter I (ID/OD 1.8/2.7 mm) was introduced over the guide wire until



Fig. 1. Teflon endoprosthesis (ID/OD 3.0/4.0 mm) with bending at varying degrees.

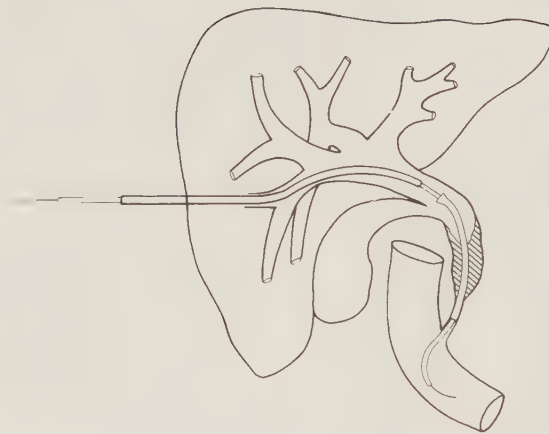
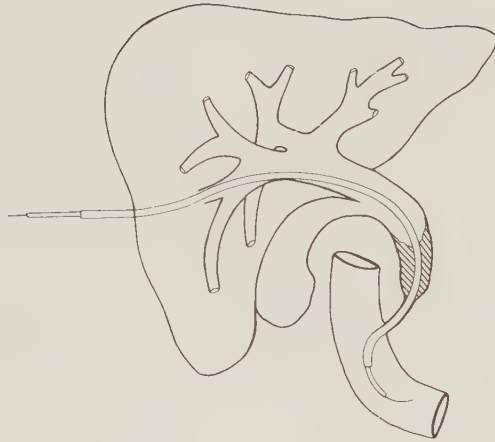


Fig. 2. Technique of percutaneous transhepatic insertion of bile duct endoprosthesis through tumor stricture.

- A. (top) Catheters I and II inserted coaxially over guide wire through tumor stricture with tip in duodenum.
- B. (bottom) Endoprosthesis and Catheter II advanced coaxially over Catheter I.

its tip was in the duodenum. Catheter II (ID/OD 3.0/4.0 mm) was introduced coaxially over Catheter I (Fig. 2 a). During this maneuver it was necessary to check that Catheter I was not pushed further into the duodenum. When the tips of both catheters were in end-to-end position in the duodenum, Catheter II was removed and re-inserted a few times. By these means a canal through the liver parenchyma and the tumor stricture was established, facilitating the insertion of the endoprosthesis. Catheter II was then removed. The endoprosthesis was mounted on Catheter I, which remained with its tip in the duodenum. The endoprosthesis was moved by hand, gliding coaxially over Catheter I until its proximal (= flared) end reached the skin of the patient. Thereupon Catheter II was remounted on Catheter I behind the endoprosthesis and applied to push the endoprosthesis into the intended position (Fig. 2 b.). The maneuver was performed under fluoroscopic control. Care was taken not to place the endoprosthesis further distally into the bile duct than planned. Catheters I and II were removed, with the special guide wire still in place in the duodenum. The procedure was terminated by inserting a polyethylene catheter (ID/OD 1.4/2.2 mm) over the guide wire with its tapered tip in the proximal part of the endoprosthesis. Side-holes close to the tip of the catheter facilitated flushing of the endoprosthesis with saline and injection of contrast medium for a check of its patency. The guide wire was removed and the catheter affixed to the skin by a suture. This catheter was finally removed after a few days, when adequate drainage via the endoprosthesis was demonstrated by injection of contrast medium. In cases with non-well-functioning endoprosthesis the catheter was left in place for external bile drainage.

RESULTS

At PTC the site of obstruction was found to be localized in the common bile duct in eight cases (Fig. 3), in the common hepatic bile duct in two cases, and in the confluence of the hepatic ducts in two cases. A stricture of the confluence of the hepatic ducts and, in addition, a stricture of the common bile duct were found in one case (Fig. 4). The approximate length of the bile duct stricture/occlusion was between 2 and 6 cm (mean 4 cm). The length of the endoprosthesis varied between 4 and 12 cm (mean 8 cm). The proximal orifice of the endoprosthesis was in the right hepatic duct in four patients, whereof three had tumor occlusion of the confluence of the hepatic ducts. The duct system of the left liver lobe was not drained via the endoprosthesis in these cases. The proximal orifice of the endoprosthesis was in the common hepatic or common bile duct in nine patients. The endoprosthesis was placed with its distal orifice in the distal segment of the common bile duct in ten cases and in the duodenum in three cases. The duodenal segment of the endoprosthesis was approximately 1.5 cm long.



Fig. 3. 53-year-old female with periampullary carcinoma and multiple liver metastases. Stricture of proximal segment of common bile duct. 12-cm-long endoprosthesis () in common hepatic and common bile ducts. Slightly dilated intrahepatic bile ducts. Contrast medium injected through percutaneous transhepatic catheter passes through endoprosthesis into duodenum.



Fig. 4. 62-year-old female previously operated on because of carcinoma of gallbladder. Metastases in porta hepatis (silver clips) and hepatoduodenal ligament. Stricture of confluence of hepatic ducts and proximal segment of common bile duct. 10-cm-long endoprosthesis with proximal orifice () in right hepatic ducts. No drainage of bile ducts of left liver lobe. Injection of contrast medium via percutaneous transhepatic catheter demonstrates grossly dilated intrahepatic bile ducts. Distal orifice of endoprosthesis () directed against wall of common bile duct resulting in functional disorder.

Spontaneous dislocation of the endoprosthesis occurred in five patients. In one case the endoprosthesis was observed to be dislodged nearly completely into the duodenum eight days after its insertion. Via a simultaneous indwelling percutaneous transhepatic catheter the endoprosthesis was pushed over a guide wire into the duodenum and a second endoprosthesis was placed in the distal segment of the common bile duct. The first endoprosthesis passed without complication through the digestive tract. The second glided about 1 cm through the papilla and has remained in this position for eight months. In two more cases the endoprosthesis glided in distal direction so that 1 - 2 cm came within the duodenum. No efforts were made to correct the position of the endoprosthesis because the drainage function was not impaired. In one patient the endoprosthesis glided about five days after insertion from the distal segment of the common bile duct in proximal direction and was detected in the confluence of the hepatic ducts. No effort was made to retrieve it. Bile drainage was attained via a simultaneously placed percutaneous transhepatic catheter until the patient died because of tumor cachexia two weeks later. In one patient the endoprosthesis rotated 180° in its position in the distal segment of the common bile duct, resulting in functional occlusion of its distal orifice. Permanent external bile drainage was facilitated through a percutaneous transhepatic catheter.

The endoprosthesis remained in the intended position in eight cases. In three of these Bucrylat (Isobuthyl-2-cyanoacrylate, Ethicon GmbH, Germany) was injected percutaneously around the midpoint of the endoprosthesis through a fine needle (OD 0.9 mm) guided by biplane fluoroscopy. The intention was to attach the endoprosthesis to the surrounding tissue by the adhesive material. The endoprosthesis was occluded inadvertently by Bucrylat in one case and permanent palliative external bile drainage had to be carried out via a percutaneous transhepatic catheter. Nine of totally 13 patients had an endoprosthesis between eight days and five months prior to death. The remaining four patients are alive five, six, and eight weeks, and eight months, respectively, after receiving a bile duct endoprosthesis.

All patients had a percutaneous transhepatic catheter for flushing after insertion of the endoprosthesis. This catheter was removed in seven patients after four days to three weeks. Subsequently the bile was drained via the endoprosthesis only. The remaining six patients had a permanent transhepatic catheter because of insufficient bile drainage through the endoprosthesis. In three of these cases the endoprosthesis was inserted in a position with either the distal or the proximal (Figs. 4 and 5) orifice pointing against the wall of the bile duct. No bile flow through the tube was possible. The endoprosthesis was occluded by Bucrylat in one case. Spontaneous dislocation causing functional disorder of the endoprosthesis occurred in two cases. In patients with bile drainage exclusively through the bile duct endoprosthesis (Group I) the drainage effect was estimated at the levels of total serum bilirubin and serum alkaline phos-



Fig. 5. 88-year-old male with periampullary carcinoma occluding distal segment of common bile duct. 5-cm-long endoprosthesis with proximal orifice () directed against wall of common bile duct. Injection of contrast medium through percutaneous trans-hepatic catheter demonstrates dilated intra- and extrahepatic bile ducts. No flow of contrast medium into duodenum.



Fig. 6. 22-year-old female with chronic inflammatory mass in porta hepatis and hepatoduodenal ligament (Crohn's disease).

- A. Intrahepatic and common hepatic ducts grossly dilated. Common bile duct strictured in its total length. (Reprinted with the permission of Georg Thieme Publishers, Stuttgart: from *Fortschr Röntgenstr* 129:533 - 550, 1978).



Fig. 6.B. Endoprosthesis in common bile duct with tip in duodenum. Injection of contrast medium through percutaneous transhepatic catheter demonstrates marked regress of bile duct dilatation and flow of contrast medium through endoprosthesis into duodenum.



Fig. 6.C. Bile duct endoprosthesis in same position five months later.
Gas in slightly dilated intrahepatic bile ducts.

phatase. In patients with an endoprosthesis and a percutaneous transhepatic catheter (Group II) the amount of externally drained bile was considered in addition. These two groups were evaluated separately.

Group I: Bile duct endoprosthesis

Seven patients had a bile duct endoprosthesis during a period ranging from one to 35 weeks. Four of these patients died one, two, eight, and 24 weeks after they had received the endoprosthesis. Three patients are alive and have had an endoprosthesis for five, six, and 35 weeks, respectively (Fig. 6). In no case were the total serum bilirubin and the serum alkaline phosphatase reduced to normal values (Table I). The values for total serum bilirubin were unchanged in two patients and had decreased slightly to markedly in four patients. In one patient the total serum bilirubin value was moderately increased with minor fluctuations throughout the drainage period.

Group II: Bile duct endoprosthesis and percutaneous transhepatic drainage catheter

Six patients had an endoprosthesis and a percutaneous transhepatic drainage catheter simultaneously during a period ranging from two to eight weeks (Table II). Five of these died two, three, three, five, and five weeks after receiving the endoprosthesis. One patient is alive and has had an endoprosthesis for eight weeks. The total serum bilirubin level decreased to normal values in one case and was reduced markedly in two. In two patients the total serum bilirubin values were unchanged during the drainage period. In one patient the total serum bilirubin increased markedly. The volume of externally drained bile varied between 150 and 800 ml/24 hours. There was no relation between the levels of the total serum bilirubin/alkaline phosphatase and the amount of externally drained bile.

TABLE I. Bile drainage via endoprosthesis

Drainage period	Total bilirubin serum level during period of drainage (normal range 3-20 $\mu\text{mol/l}$)	Alk.phosph. serum level during period of drainage (normal range 0.8 - 4.6 $\mu\text{kat/l}$)
1 week, dead	100, unchanged	8, unchanged
2 weeks, dead	60, unchanged	8, unchanged
5 weeks, alive	100 $\rightarrow$ 75	14, unchanged
8 weeks, dead	100 $\rightarrow$ 27	21 $\rightarrow$ 40 $\rightarrow$ 25
6 weeks, alive	305 $\rightarrow$ 150	10 $\rightarrow$ 21
24 weeks, dead	224 $\rightarrow$ 130 $\rightarrow$ (190)	12 $\rightarrow$ 16 $\rightarrow$ 56
35 weeks, alive	41 $\rightarrow$ (80) $\rightarrow$ 35	9 $\rightarrow$ 10 $\rightarrow$ 9

TABLE II. Bile drainage via percutaneous transhepatic catheter. Functional disorder of endoprosthesis

Drainage period	Total bilirubin serum level during period of drainage (normal range 3-20μmol/l)	Alk.phosph.serum level during period of drainage (normal range 0.8 - 4.6 μkat/l)	Total volume of bile drained externally during 24 hours
2 weeks, dead	70 unchanged	17, unchanged	300 - 500 ml
3 weeks, dead	160 → 50	10 → 7	500 - 800
3 weeks, dead	120 → 60	23 → 15	150 - 200
5 weeks, dead	62 → 158	20, unchanged	400 - 600
5 weeks, dead	37 unchanged	11, unchanged	300 - 500
8 weeks, alive	110 → 12	26 → 9	300 - 600

COMPLICATIONS

Formation of blood clots within the hepatic ducts in connection with the placement of a percutaneous transhepatic catheter, was observed in one patient without clinical symptoms of hemobilia. The injection of contrast medium via the transhepatic bile duct catheter six days later demonstrated dissolution of the clots. One patient drained blood through the percutaneous transhepatic catheter 10 days after insertion of the bile duct endoprosthesis. The external bleeding continued after removal of the transhepatic catheter. At angiography a 2 x 3 cm aneurysm was observed in the central part of the right liver lobe. The portal vein was shown to be patent. The general condition of the patient excluded surgical intervention. In order to stop the otherwise uncontrollable hemorrhage, gel foam particles were injected into the common hepatic artery, which originated from the superior mesenteric artery. The aneurysm was partially occluded together with most of the segmental arteries of the liver. The bleeding ceased after this procedure. Three days later the patient developed liver failure and died. Post-mortem examination revealed marked hepatic infarcts and extensive necroses of the liver.

Prior to insertion of a bile duct endoprosthesis four patient with a percutaneous transhepatic drainage catheter during a period longer than three weeks developed cholangitis, which responded well to antibiotic therapy. In three additional patients without clinical symptoms of cholangitis, cultures verified infection of the bile drained through the percutaneous transhepatic catheter. Four patients experienced tenderness below the right costal arch and spiky fever one to three days following insertion of the endoprosthesis. Septicemia was verified by blood cultures in one of these cases. In all patients the cholangitis and sepsis were effectively treated by antibiotic therapy. *Staphylococcus aureus* and *Enterobacteria* were isolated in cultures from the bile drained through the indwelling percutaneous transhepatic catheter following the introduction of an endoprosthesis in six patients who did not present with clinical signs of cholangitis.

Incorrect placement of the endoprosthesis, resulting in functional occlusion of its proximal or distal orifice by the wall of the bile ducts, occurred in three patients. Spontaneous dislocation of the endoprosthesis caused ineffective bile drainage in two patients whereas minor dislodgement (i.e. less than 2 cm) in three cases did not hinder the bile passage through the teflon tube.

FINDINGS AT AUTOPSY

Post-mortem examination was carried out in six of nine patients who had a bile duct endoprosthesis between two and 24 weeks prior to death. The endoprosthesis was patent in four cases (Fig. 7) after two, three, and eight weeks, respectively. The bile ducts were slightly (three cases) or moderately (one case) dilated. The formation of an aneurysm in the right lobe of the liver with severe hemorrhage through the percutaneous transhepatic catheter and marked hemobilia, resulted in occlusion of the bile duct endoprosthesis by blood clots in one case. Complete obliteration of the endoprosthesis by inspissated bile and cell debris was found in one patient (Fig. 8) five months after insertion of the teflon tube. Suppurative cholangitis was present in two patients. In one the endoprosthesis drained the right liver lobe only. The ducts of the left liver lobe were grossly dilated, containing purulent bile (Fig. 4). The other patient had multiple abscesses combined with purulent cholangitis in both liver lobes.

DISCUSSION

When obstruction of the extrahepatic bile ducts by a malignant tumor is suspected, percutaneous fine-needle aspiration biopsies for cytodiagnosis verify the diagnosis in many cases (30). Furthermore, arteriography and percutaneous transhepatic portography may contribute essentially to the evaluation of tumor resectability before operation (31 - 34). Exploratory laparotomy for diagnostic purposes may thus be omitted when the local extent of the disease is too far advanced to perform radical surgery. Palliative biliary drainage procedures should, however, be considered, to relieve jaundice and pruritus.

There is continuing controversy in the literature as to the risks of bypass surgery in obstruction of the extrahepatic bile ducts because of malignant tumors. Crile (35) concluded that patients with symptomatic carcinoma of the head of the pancreas who underwent by-pass procedures without resection have a longer survival than those radically operated. Moertel (36) has shown that biliary diversion combined with gastroenterostomy can result in increased survival. Several authors, however, report that surgical decompression of extrahepatic cholestasis due to malignant tumors may imply a high risk of operative mortality (37 - 39). Coutsoftides (40) found that patients undergoing by-pass did not live longer than those undergoing exploratory laparotomy.



Fig. 7. 83-year-old female with carcinoma involving distal segment of common hepatic and proximal segment of common bile duct. At autopsy, endoprosthesis patent. Slight dilatation of bile ducts proximal to tumor stricture (➡).

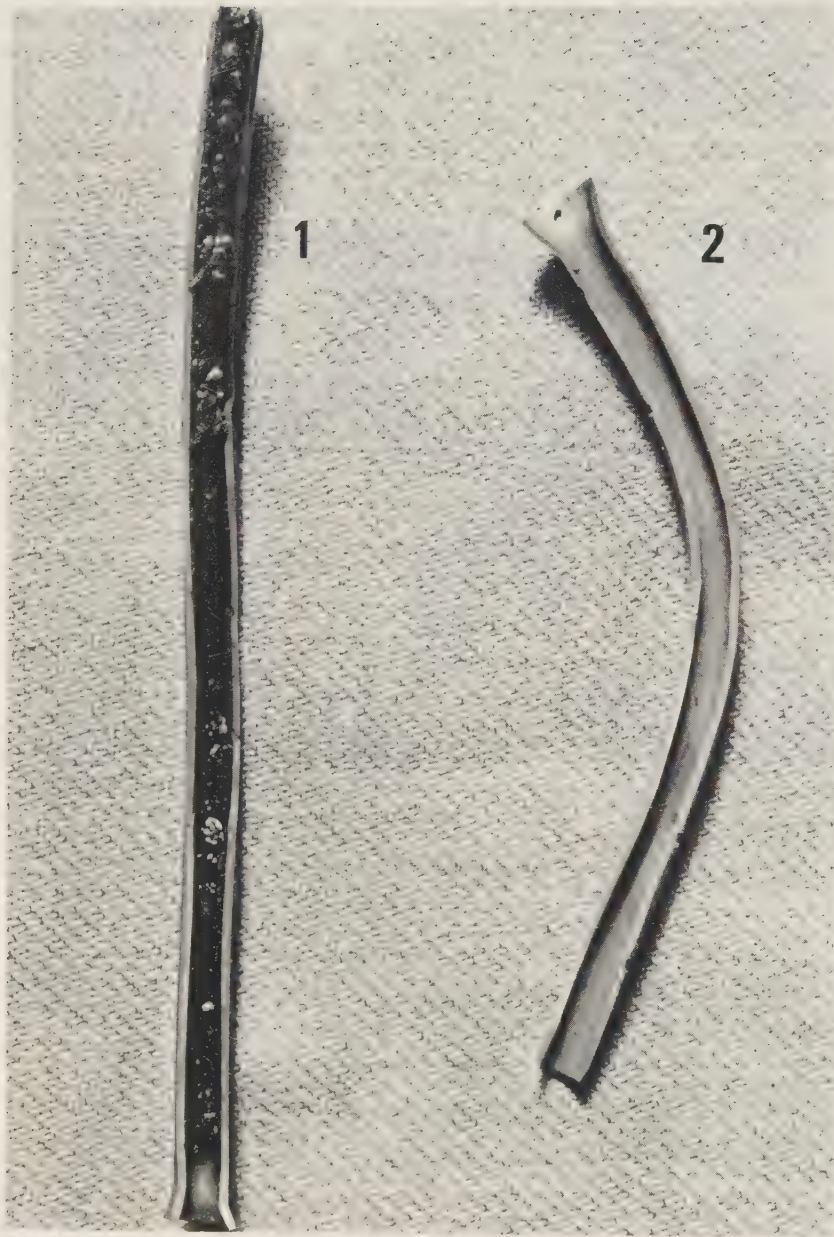


Fig. 8. Endoprosthesis (1) obliterated by inspissated bile and cell debris after drainage period of five months in patient with perampullary carcinoma. Endoprosthesis (2) patent after drainage period of two months in patient with carcinoma of gallbladder and tumor infiltration of hepatoduodenal ligament.

In selected patients with malignant obstruction of the extrahepatic bile ducts, a non-surgically inserted bile duct endoprosthesis may be an alternative. The results of the present investigation on the percutaneous transhepatic insertion of a bile duct endoprosthesis are discussed and compared with the experiences of Burcharth (26, 27) and Pereiras (28) who are the only authors reporting the non-surgical insertion of a bile duct endoprosthesis in larger groups of patients. Burcharth used a 15-cm long polyethylene catheter (ID/OD 1.5/2.0 mm) with side-holes in 48 patients with extrahepatic cholestasis caused by a malignant tumor (27). Jaundice disappeared in approximately 65 per cent of his patients who obtained a permanent endoprosthesis. The median survival time in a previous report (26) on 17 patients was 61 days (range 24 - 427 days). In all of these patients jaundice recurred approximately one month before death. Dislocation of the endoprosthesis occurred in one patient.

Pereiras et coll (28) reported the insertion of a teflon tube without side-holes in 12 patients with unresectable neoplasms. The endoprosthesis which he applied had the same diameter (ID/OD 3.0/4.0 mm) as the endoprosthesis used in the present investigation. In 10 of 12 patients the serum bilirubin values normalized or decreased markedly and jaundice disappeared. The longest survival time was 31 weeks. Dislocation of the endoprosthesis, cholangitis, and bleeding did not occur. One patient developed a biliary pleural effusion two days after the procedure which was drained by thoracocentesis and never reaccumulated. Six patients had transient fever 24 to 48 hours after the procedure. Percutaneous transhepatic introduction of an endoprosthesis has been performed by Wiechel (25) but no detailed report is available.

The complication rate and functioning of the endoprosthesis in the cases described herein differ markedly from those reported by Burcharth (26, 27) and Pereiras (28). One patient died because of fatal hemorrhage from an aneurysm in the liver which was not present prior to the percutaneous transhepatic intubation, as proved by angiography. Whether this damage was caused by insertion of the percutaneous transhepatic catheter or occurred in connection with the subsequent placement of the endoprosthesis, it must be attributed to the method. Aneurysms of intrahepatic arteries after diagnostic percutaneous transhepatic cholangiography and needle biopsy of the liver have previously been demonstrated by angiography (41 - 43). Whereas Burcharth (26, 27) used as endoprosthesis a thin polyethylene catheter with an outer diameter of 2.0 mm, the teflon tube applied in the present study had an outer diameter of 4.0 mm. The proximal end was flared to prevent dislocation of the teflon tube. By that means the placement of the endoprosthesis resulted in a broad track through the liver parenchyma. This canal was demonstrated to be patent in one case 10 days after insertion of the endoprosthesis (Fig. 9). Mechanical trauma is most likely the main reason for the formation of intrahepatic aneurysms as well as other vascular complications following needle puncture of the liver (41 - 44). Infection of the puncture canal through the liver,



Fig. 9. 77-year-old female with endoprosthesis because of carcinoma of proximal segment of common bile duct. Injection of contrast medium through fine needle into moderately dilated intrahepatic bile ducts demonstrates flow through endoprosthesis into duodenum. Track through liver parenchyma following insertion of endoprosthesis filled with contrast medium ().

TABLE III. Reason for functional disorder of bile duct endoprosthesis

Primarily unfavorable position of endoprosthesis	3 cases
Spontaneous dislocation of endoprosthesis	2 cases
Occlusion of endoprosthesis by debris and inspissated bile	1 case ^{x/}
Occlusion of endoprosthesis by tissue adhesive agent	1 case

x/ This patient had no percutaneous transhepatic drainage catheter in addition to the endoprosthesis.

however, may additionally favor the formation of an aneurysm. Bile duct infection was present in three cases of aneurysm formation reported in a study on bile drainage via percutaneous transhepatic catheter (19). An aneurysm adjacent to the transhepatic drainage tube is not suspected unless hemorrhage occurs. Angiography is therefore highly recommendable when the placement of a bile duct endoprosthesis is considered following drainage through a percutaneous transhepatic catheter. When the puncture is made from the right midaxillary line, the risk of arterial vascular complications can be reduced by intubating the biliary system from a peripheral bile duct of the right lobe of the liver. By these means the drainage catheter passes the liver parenchyma intraductally to the greatest possible extent and the risk of mechanical damage to the segmental hepatic arteries in the central area of the liver is diminished.

A major problem in bile drainage through a percutaneous transhepatic catheter is the high frequency of infection of the biliary tract. The incidence of cholangitis in patients with percutaneous transhepatic bile drainage was analyzed in a recent investigation (15). Cholangitis was evident in about 25 per cent of all patients with a drainage catheter during a period ranging from one day to 19 months. Almost all patients with prolonged drainage were reported to have at least one attack of cholangitis. Burcharth (27), however, reported only two cases of cholangitis among 48 patients and Pereiras et coll (28) found no cholangitis in 12 patients with bile duct endoprosthesis. The risk of bile duct infection is apparently correlated to the length of the drainage period before an endoprosthesis is inserted. Burcharth (26) placed the endoprosthesis immediately following the diagnostic percutaneous transhepatic cholangiography and removed the external drainage catheter within three days. The patients in the present material were drained through a percutaneous transhepatic catheter during a period ranging between eight days and nine months before they finally received an endoprosthesis. During this period infection of the biliary tract was proven in seven of 13 patients by clinically symptomatic cholangitis and/or isolation of bacteriae from the bile drained through the percutaneous transhepatic catheter.

The efficiency of the endoprosthesis depended on its position within the bile duct, its patency, and the viscosity of the bile. We found that primarily poor insertion (3 cases) and spontaneous dislocation of the endoprosthesis may result in insufficient drainage function or no function at all (2 cases). Renewed percutaneous transhepatic access to the bile duct system may render possible the insertion of a new endoprosthesis. This was successfully performed in one of the patients reported here. Subsequent bile drainage depended exclusively on a percutaneous transhepatic catheter in two patients with insufficient drainage due to dislocation of the endoprosthesis (Table III). In three patients with tumors at the confluence of the hepatic ducts, introduction of an endoprosthesis resulted in non-drainage of the left liver lobe. The risk of purulent cholangitis of the occluded duct system of the left liver lobe was regarded

as high but nevertheless acceptable because of the palliative nature of the method in patients in whom surgery was deemed not feasible.

The functioning of the endoprosthesis was regarded as partially effective in seven patients. In two of these with an endoprosthesis for one and two weeks only, the total bilirubin serum level was raised but did not further increase. In the remaining five patients the serum bilirubin values decreased slightly to markedly, whereas the serum alkaline phosphatase level was unchanged or increased moderately. After five months of sufficient drainage function in one patient the serum bilirubin and alkaline phosphatase levels increased rapidly one week prior to death. At autopsy, purulent cholangitis and occlusion of the endoprosthesis were found (Fig. 8). Optimal bile drainage through the endoprosthesis should result in normalizing of the serum bilirubin and alkaline phosphatase levels, if cholestasis is caused by extrahepatic bile duct occlusion only. Technical shortcomings such as the design of the endoprosthesis and/or its final position in the extrahepatic ducts must therefore be considered the main reasons for functional disorder.

The type of endoprosthesis applied in this study was designed without side-holes to minimize occlusion by debris from the wall of the bile duct adjacent to the teflon tube. In four patients the endoprosthesis was proved to be open at autopsy, whereas the teflon tube was obstructed in its entire length in a case of purulent cholangitis. Lack of side-holes, however, resulted in a functional disorder whenever the proximal or distal opening of the endoprosthesis was directed against the wall of the bile duct (Figs. 4, 5).

Burcharth (26, 27) used fine polyethylene endoprostheses with side-holes and observed good drainage function in 65 per cent of patients who had a permanent endoprosthesis. It is, however, rational to assume that tubes with wider diameter provide better bile passage than do fine tubes. Furthermore, the risk of occlusion by cell debris and inspissated bile should be less than with a fine tube. This is confirmed by experience with operative insertion of palliative T- and U-tube drainage in carcinoma of the main hepatic duct junction, which has shown that even large-bore tubes may occlude in time (45).

The optimal type of endoprosthesis is not yet available. It appears from the experience of the present authors and that of Burcharth (26, 27) that side-holes in both the proximal and distal parts of the endoprosthesis seem to be essential for sufficient drainage function. Furthermore there is no reason to believe that finer catheters and endoprostheses which

have been applied to drain bile externally (13, 15), combined internally/externally (16 - 22, 24), and internally (26, 27), are less traumatic than large-bore tubes. The formation of intrahepatic aneurysms following bile duct intubation with a percutaneous transhepatic catheter (OD 2.2 mm) has been described (13, 19).

Based on the results of the present study and those reported by Burcharth (26, 27) and Pereiras et coll (28), a non-surgical bile duct endoprosthesis may be used as a palliative procedure. When the preoperative evaluation has shown a cytologically verified malignant tumor obstructing the extrahepatic bile ducts, several groups of patients may be considered for permanent palliative non-surgical bile duct drainage via an endoprosthesis:

1. Elderly patients in poor general condition,
2. Patients with generalized malignant disease,
3. Patients with metastatic tumor obstructing the extrahepatic bile ducts,
4. Patients not belonging to groups 1 to 3, in whom an operative bilio-digestive anastomosis is not feasible for technical reasons.

In patients with an angiographically demonstrated intrahepatic aneurysm adjacent to a percutaneous transhepatic catheter, however, non-surgical insertion of an endoprosthesis is contraindicated.

It is our present opinion that a satisfactorily functioning endoprosthesis is preferable to bile drainage via a percutaneous transhepatic catheter indwelling in the bile ducts. We advocate that patients not belonging to groups 1 to 4 should be operated upon with a bilio-digestive anastomosis. In patients with unresectable carcinoma of the hepatic confluence, the U-tube drainage described by Terblanche et coll (45) and a comparable method described by Praderi (46) may be considered. The great advantage of their methods is that the tube is replaceable in case of occlusion without laparotomy.

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A Comparative Diagnostic Study of Malignant Lesions of the Liver by Infusion Angiography and Percutaneous Transhepatic Portography

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11 Figures

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The diagnostic effectiveness of percutaneous transhepatic portography (PTP) was evaluated in 21 patients with primary and metastatic liver carcinoma, who also had infusion hepatic angiography. The findings at PTP were compared with unequivocal tumor changes demonstrated at infusion hepatic angiography in all cases. PTP revealed tumors in 14 of 21 cases. The location, size and number of the tumor(s) were decisive for their detectability at PTP. *Solitary* lesions smaller than 2 cm in the central parts of the liver escaped detection, whereas lesions larger than 7 cm in the left lobe and 6 cm in the right lobe were demonstrated in all cases. *Multiple* metastases of 1–2 cm were detected in most cases. Lesions smaller than 4 cm were better shown by the sinusoidal phase at PTP, whereas lesions larger than 5 cm were better delineated by the portal venous phase.

Infusionsangiographie und perkutane transhepatische Portographie bei malignen Lebertumoren. Eine vergleichende diagnostische Studie

Die Leistungsfähigkeit der perkutanen transhepatischen Portographie (PTP) als diagnostische Methode wurde bei 21 Patienten mit primären und metastatischen Leberkarzinomen untersucht. Bei allen Patienten wurde zusätzlich eine Infusionsangiographie der Leber durchgeführt. Die Ergebnisse der PTP wurden mit Befunden der Infusionsangiographie verglichen, die raumfordernde Prozesse in allen Fällen zweifelsfrei erkennen ließen. Bei 14 von 21 Patienten wurde mit der PTP ein raumfordernder Prozeß nachgewiesen. Die Erkennbarkeit der Tumoren bei der PTP war abhängig von ihrer Lage, Größe und Anzahl. In den zentralen Anteilen der Leber konnten kleine (< 2 cm), *solitäre* Tumoren nicht nachgewiesen werden. Im rechten Leberlappen konnten Tumoren über 6 cm und im linken Leberlappen Tumoren über 7 cm Größe in allen Fällen nachgewiesen werden. *Multiple*, kleine (1–2 cm) Metastasen wurden in den meisten Fällen diagnostiziert. Tumoren in der Größenordnung unter 4 cm wurden besser in der Sinusoid-Phase der PTP dargestellt, während Tumoren über 5 cm Größe besser in der Venen-Phase nachgewiesen werden konnten.

Ever since *Bierman* et al. (1951) (2, 3) published their results of intra-arterial catheterization of viscera in man, numerous investigators have introduced and refined more sophisticated angiographic techniques rendering possible detailed studies of the arterial and venous systems of the liver. *Boijesen* (1965) (4), advocated the selective catheterization of the hepatic artery (-ies) as providing more detailed information from the late arterial and early capillary phase, as superior to celiac and mesenteric angiography. *Wise* et al. (1968) (20) further improved the diagnostic yield of liver angiography when performing "infusion" studies (i.e. 75 ml contrast medium at a flow rate of 2 ml/sec via a catheter surgically placed in the hepatic artery primarily for selective cytotoxic therapy). *Kaude* et al. (1973) (11) and *Freeny* et al. (1976) (6) evaluated the effectiveness of infusion angiography of the liver in comparison with conventional hepatic angiography. At conventional angiography 20–50 ml contrast medium was injected at a flow rate of 5–12 ml/sec. Infusion angiography was performed with 30–50 ml contrast medium at a flow rate of 2.5–5 ml/sec. *Kaude* et al. (1973) (11) found the infusion technique superior to conventional hepatic angiography in 14 out of 22 patients with metastatic liver tumors. In 8 cases it gave equal information, and in no case was it less informative than the conventional technique. *Freeny* et al. (1976) (6) injected 36–60 ml contrast medium at a flow rate of 3–5 ml/sec selectively into



Fig. 1 38-year-old male. PTP with tip of catheter in main stem of portal vein. Normal distribution of portal venous ramification.

Figs 2A–C 63-year-old female with multiple liver metastases in both liver lobes. Previously operated on because of gastric carcinoma.



Fig. 2A Infusion hepatic angiography: Multiple angiographically avascular metastases in right liver lobe. No satisfactory demonstration of left liver lobe.



Fig. 2B PTP – portal venous phase: Multiple areas with occlusion of minor intrahepatic portal vein branches in both liver lobes. Partial compression of main stem of portal vein due to space – occupying lesion in porta hepatis.



Fig. 2C PTP – sinusoidal phase: Multiple areas of reduced contrast medium accumulation in both liver lobes due to metastases.

Table I Site and type of primary tumors in 21 patients

Primary tumor	No. of cases
Carcinoma of thyroid	1
Carcinoma of kidney	1
Carcinoma of pancreas	2
Carcinoma of stomach	1
Carcinoma of colon	5
Carcinoid of small bowel	4
Melanoma of skin	1
Primary carcinoma of liver	6
TOTAL	21

the hepatic artery in 45 patients with malignant liver tumors. In comparison with the conventional technique the infusion studies gave essential and improved diagnostic information in 82% of cases whereas they were classified as non-contributory to the diagnosis in 18%. *Wirtanen* (1973) (19) examined 332 patients with primary and secondary liver carcinoma via infusion angiography (50 ml contrast medium injected selectively into the hepatic artery at a flow rate of 5 ml/sec) and observed accumulation of contrast medium in the tumor area in 97.5% of cases.

The aim of this study was the evaluation of the diagnostic effectiveness of percutaneous transhepatic portography (PTP) in primary malignant lesions and metastases of the liver. It was regarded as essential that infusion hepatic angiography, a highly reliable diagnostic method, be performed within a short lapse of time prior to PTP. The findings at PTP were compared only with unequivocal tumor changes demonstrated at infusion hepatic angiography. No claim is made that all malignant lesions present were demonstrated by infusion angiography.

Material

The series comprised 21 patients: 12 women and 9 men ranging from 26 to 77 years of age. Suspicion of metastases to the liver in patients with known primary tumors (15 cases) or suspicion of primary carcinoma of the liver (6 cases) was the indication for angiographic studies. PTP was performed the same day or after a delay of up to 10 days (mean 4 days). The final diagnosis of the primary tumor and liver metastases was verified by fine-needle aspiration biopsy, resection, or autopsy in all cases. The site and type of the primary tumors are given in Table I. In addition to primary liver carcinoma, cirrhotic changes of the liver were present in 3 patients. Liver cirrhosis was not found in patients with metastases to the liver.

Methods

Prior to PTP and arteriography all patients were premedicated with 10 mg diazepam and 0.5 mg atropine intramuscularly. The arteriographic studies were carried out via the transfemoral route in 20 patients and via the left transbrachial access in one patient.

Figs 3A–C 67-year-old female with two metastases in right liver lobe. Previously operated on because of carcinoma of colon.

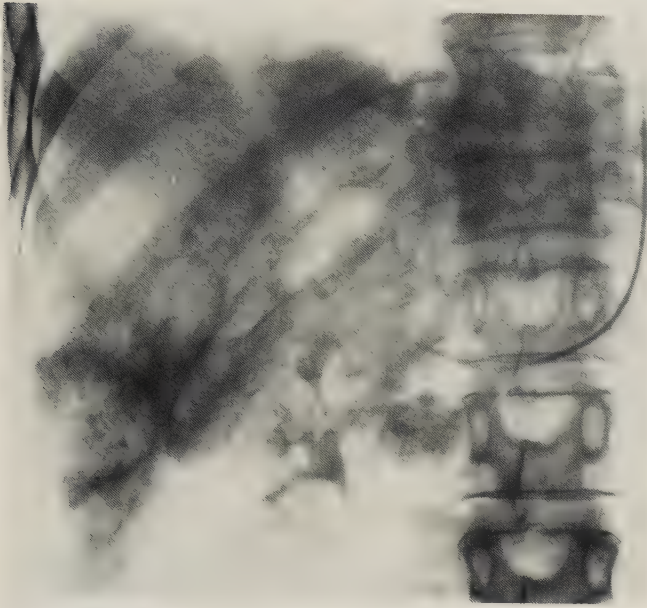


Fig. 3A Infusion hepatic angiography: Two hypovascular metastases with hypervascular periphery (4.5 and 3 cm) in lower part of right liver lobe.



Fig. 3B PTP – portal venous phase: About 4 x 3 cm area with occlusion of minor portal vein branches (→) in lower part of right liver lobe. Slight dislocation of larger intrahepatic portal vein branch (↗).

Polyethylene catheters (transfemoral: ID/OD 1.4/2.2 mm, transbrachial: ID/OD 1.16/1.6 mm) preshaped by hand were used throughout. Prior to the hepatic infusion study, selective celiac (40 ml contrast medium/flow rate 10 ml/sec) and superior mesenteric angiography were performed. As patency of the portal vein was a prerequisite for PTP, the study of the superior mesenteric artery was made following the injection of 25 mg tolazoline hydrochloride diluted in 10 ml saline into the superior mesenteric artery (60 ml contrast medium/flow rate 12 ml/sec). This examination was made with the patient in the left anterior oblique position and resulted in visualization of the main stem of the superior mesenteric vein and the portal vein in all cases.

For the infusion studies the catheter was placed in the common hepatic artery in 12 cases and in the proper hepatic artery in 7 cases. In 2 cases catheterization was carried out of the right hepatic artery which originated from the superior mesenteric artery. In the examination of the left liver lobe in these 2 cases infusion hepatic angiography was not performed. The left gastric artery contributed in no case to the blood supply of the left lobe of the liver. The infusion studies were performed with the patient supine and an injection of 50–60 ml contrast medium (370 mg I/ml) at a flow rate of 2–4 ml/sec. The quantity of contrast medium and the flow rate were determined by estimating the degree of hepatic arterial blood flow and the size of the liver. In addition, care was taken to minimize the risk of contrast medium reflux into the splenic artery. Filming for infusion angiography was spread over 32 seconds exposing 16 films at 2-second intervals. Only cases where the hepatic infusion study was regarded as technically optimal are included in the study.

Percutaneous transhepatic portography was performed with the patient supine. Following local anesthesia of the subcutis and intercostal tissue, a 25 cm long stylet fitted with a radiopaque polyethylene catheter* (ID/OD 1.0/1.6 mm) was inserted under fluoroscopic control from the right midaxillary line towards the area directly cranial to the approximate position of the liver hilum. After removal of the stylet the catheter was slowly withdrawn until blood could be aspirated. Following identification of a portal vein branch by injection of contrast medium, a guide wire was introduced through the catheter into the portal vein branch and manipulated into the main stem of the portal vein. Subsequently, the catheter was pushed over the guide wire into the main stem of the portal vein and the guide wire was removed. The technique of percutaneous transhepatic puncture and catheterization of the portal vein was described in detail elsewhere (8, 9). For



Fig. 3C PTP – sinusoidal phase: About 4 x 3 cm area of reduced accumulation of contrast medium in same area. PTP fails to demonstrate 3 cm metastasis.

evaluation of the intrahepatic portal vein branches and the parenchyma of the liver, the tip of the catheter was placed in the proximal part of the main stem of the portal vein. With the patient in the supine position, a series with vertical beam was taken (5 films/5 sec, 8 films/4 sec, 3 films/6 sec, 40 ml contrast medium 370 mg I/ml, flow rate 8 ml/sec: Fig. 1). In addition a series with the patient in the left anterior and right anterior positions was taken in 5 cases and one case, respectively. Lateral views with horizontal beam were taken in 3 cases. For optimal demonstration of the portal venous system and the parenchyma of the

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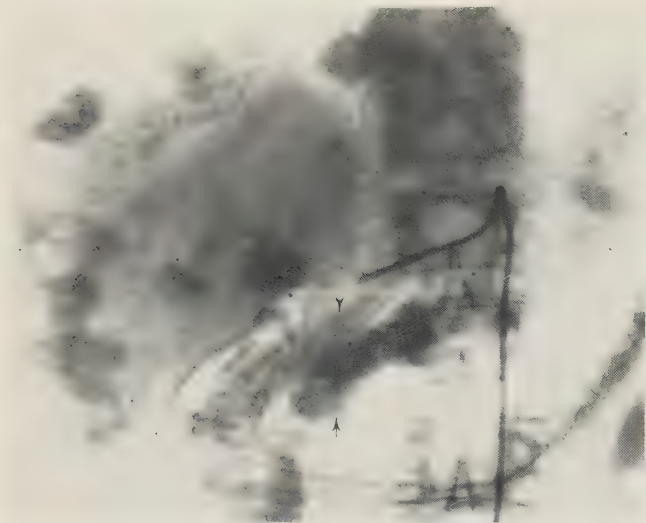


Fig. 4A

Figs 4A–E 56-year-old female with multiple metastases in both liver lobes. Previously operated on because of carcinoid of ileum.

Fig. 4A Infusion hepatic angiography: Multiple hypervascular metastases of various sizes (0.5–4.0 cm) in both liver lobes. About 4.0 × 3.0 cm metastasis in quadrate lobe (→).

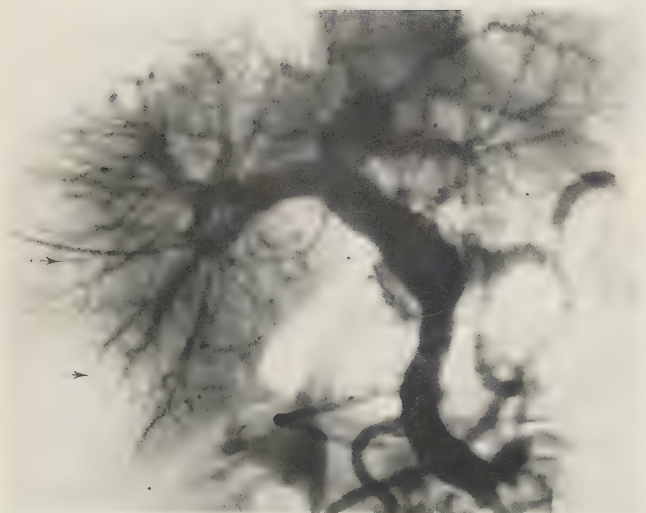


Fig. 4B

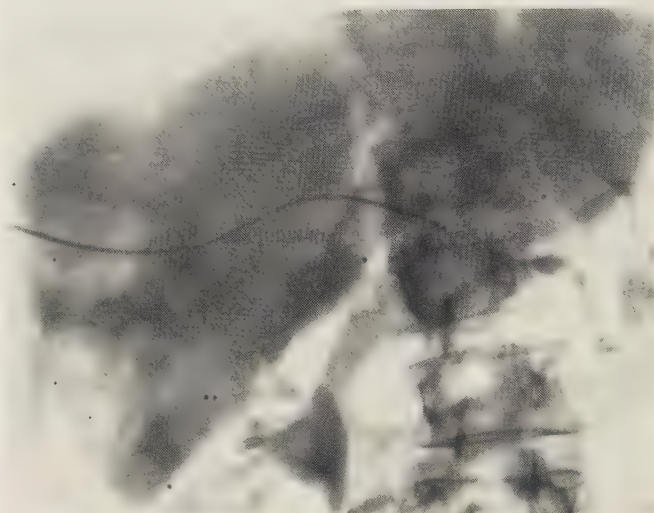


Fig. 4C

Figs 4B and C PTP – portal venous phase and sinusoidal phase: Occlusion of single peripheral portal vein branches (→). Multiple areas of reduced accumulation of contrast medium in right lobe. True degree of metastases not shown when compared with infusion hepatic study.



Fig. 4D

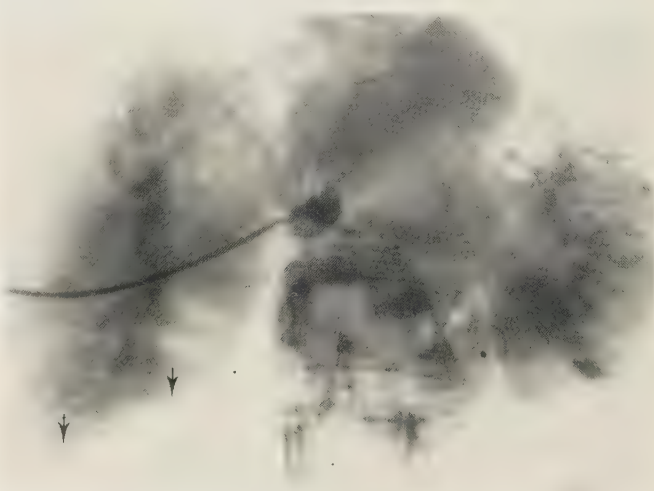


Fig. 4E

Figs 4D and E PTP – selective study of left liver lobe. Portal venous and sinusoidal phase: No metastases demonstrated in portal venous phase. Caudally in quadrate lobe, no accumulation of contrast medium in sinusoidal phase (→), possibly due to metastasis demonstrated at infusion hepatic angiography (Fig. 4A).

Figs 5A–D

26-year-old male with multiple meta-
stases in both liver lobes. Previously
operated on because of carcinoid of
ileum.



Fig. 5A

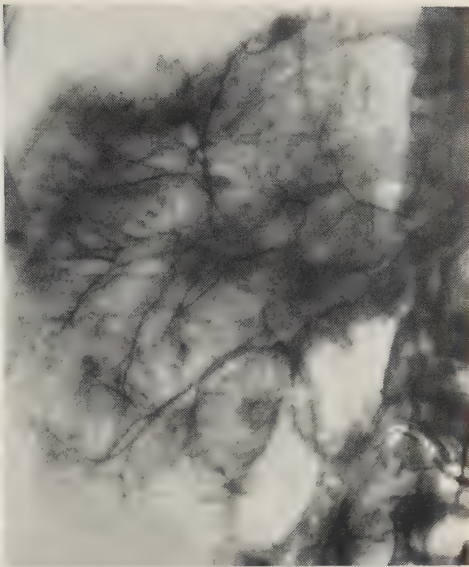


Fig. 5B

Figs 5A and B

Infusion hepatic angiography – early
and late phases: Multiple hypervas-
cular metastases of various sizes
(0.3–1.0 cm) in right liver lobe.

Table II Diagnostic information related to portal venous and sinusoidal phase of PTP in 20 cases with positive findings of space-occupying lesions.

PTP	Right liver lobe	Left liver lobe
Portal venous phase	8/12 cases	5/8 cases
Sinusoidal phase	4/12 cases	3/8 cases

Table III Correlation between size of tumor lesion demonstrated at infusion hepatic angiography and detectability at PTP

Size of lesion (cm)	Infusion hepatic angiography	PTP
< 1	1 case	0 case
1–2	7 cases	5 cases
2–6	9 cases	5 cases
6–9	2 cases	2 cases
> 9	2 cases	2 cases

left lobe, the left main branch of the portal vein was catheterized and examined selectively (20–30 ml contrast medium 370 mg I/ml, flow rate 4–7 ml/sec) in 6 cases. No complication was observed following infusion hepatic angiography or PTP in the material reported here. The technical quality of PTP was judged for each lobe as good, moderate, or poor according to the following criteria:

1. visualization of the intrahepatic portal vein branches,
2. accumulation of contrast medium in the liver parenchyma.

PTP was classified as more, equally, or less informative than infusion hepatic angiography in visualizing space-occupying lesions. In positive findings at PTP the portal venous and parenchymatous (= sinusoidal) phases were evaluated separately according to their diagnostic information. PTP was regarded as non-contributory to the diagnosis when it demonstrated tumor changes to a lesser degree than or to the same degree as infusion hepatic angiography.

Results

At infusion hepatic angiography tumor(s) were observed in both lobes in 10 cases, in the right lobe only in 7 cases, and in the left lobe only in 4 cases. In 11 patients multiple tumors



Fig. 5C



Fig. 5D

Figs 5C and D PTP – portal venous and sinusoidal phases: No metastases demonstrated. In sinusoidal phase, intrahepatic portal vein branches cleared of contrast medium appearing as areas of reduced contrast medium accumulation.

were present whereas in 10 patients a solitary tumor was found. Four solitary tumors were localized in the right and left liver lobes, respectively. In 2 cases a solitary lesion involved both liver lobes. The lesions were angiographically hypervascular in 15 cases and hypo-avascular in 6 cases. The diagnostic information was obtained from both the arterial and sinusoidal phase in 15 cases and from the sinusoidal phase alone in 6 cases.

The technical quality of PTP of the right liver lobe was good in all cases. When the contrast medium was injected into the main stem of the portal vein the visualization of the portal vein branches and the parenchyma of the left liver lobe was classified as good in 10 cases and poor in 11 cases. In 6 cases the left liver lobe was selectively investigated by injection of contrast medium into the left main branch of the portal vein, resulting in good visualization of this lobe. PTP revealed in 14 of 21 patients tumors which had been detected by infusion

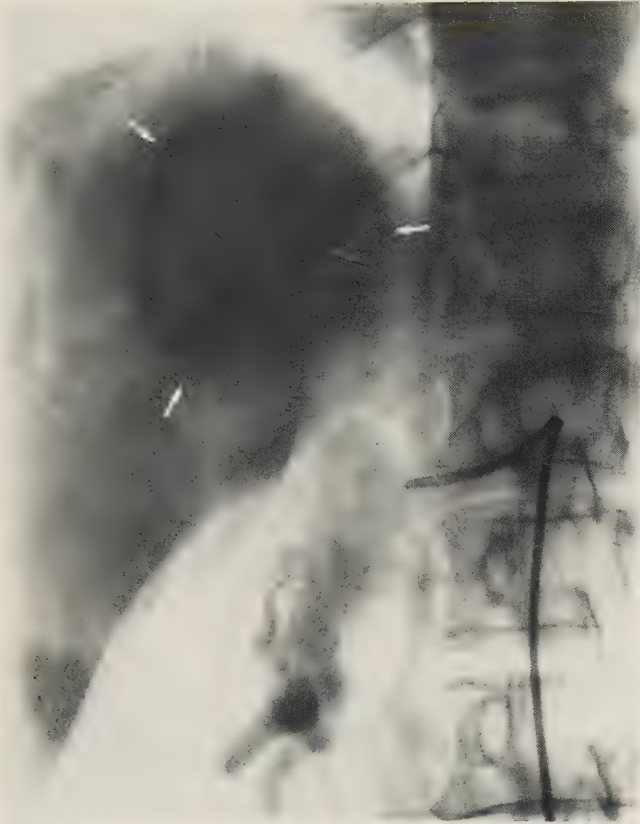


Fig. 6A Infusion hepatic angiography: Cranially in midportion of liver, increased accumulation of contrast medium (→) representing approximately 5 cm metastasis.

Figs 6A–C 58-year-old female with solitary metastasis of carcinoma of colon in caudate segment of left liver lobe infiltrating ventrocranial and dorsocaudal segments of right lobe.

Table IV Comparison of findings at infusion hepatic angiography and PTP in 21 patients with primary malignant and metastatic liver tumors.

Case	PTP		Infusion hepatic angiography	
	Number of tumors in right lobe/left lobe		Number of tumors in right lobe/left lobe	
1	multiple	solitary	multiple	multiple
2	multiple	multiple	multiple	multiple
3	multiple	multiple	multiple	multiple
4	multiple	0	multiple	multiple
5	0	0	multiple	solitary
6	multiple	solitary	multiple	solitary
7	multiple	0	multiple	multiple
8	multiple	0	multiple	x/
9	multiple	multiple	multiple	0
10	multiple	0	multiple	0
11	0	0	multiple	multiple
12	0	0	solitary	solitary
13	solitary	solitary	solitary	solitary
14	solitary	0	solitary	0
15	solitary	0	solitary	x/
16	0	0	solitary	0
17	0	0	solitary	0
18	0	solitary	0	solitary
19	0	0	0	solitary
20	0	solitary	0	solitary
21	0	0	0	solitary

0 = no space-occupying lesion observed.
x/ = left liver lobe not examined by infusion hepatic angiography.

hepatic angiography. They were found in both lobes in 6 cases, in the right lobe only in 6 cases, and in the left only in 2 cases. In 9 patients multiple lesions were demonstrated and in 5 patients a solitary lesion was detected. The solitary lesion was located in the right lobe in 2 cases and in the left lobe in 2 cases. In 1 case a solitary lesion was found to engage both lobes. In 8 of 12 cases with tumors of the right liver lobe the essential diagnostic information was obtained from the portal venous phase of the PTP, and in 4 cases from the sinusoidal phase. Tumors of the left liver lobe were detected in 5 of 8 cases in the portal venous phase and in 3 cases only in the sinusoidal phase (Table II). The detectability of space-occupying lesions by PTP was dependent on their size, number and location. Whereas all tumors beyond a size of 7 cm and most multiple metastases (1–2 cm) were demonstrated, solitary lesions smaller than 2 cm located in the central parts of the liver escaped detection (Table III). A comparative survey of the findings at PTP and infusion hepatic angiography appears in Table IV. In tumor lesions of the right liver lobe PTP provided approximately the same diagnostic information as infusion hepatic angiography in 6 of 17 cases (Fig. 2). In 6 cases (Figs. 3, 4) the number of metastases was shown to a lesser degree. In 5 cases (Figs. 5, 6) PTP resulted in falsely negative findings.

In tumor lesions of the left lobe, PTP in 3 cases provided the same diagnostic information as infusion hepatic angiography regarding the extent of the space-occupying lesions. In one case (Fig. 2) PTP showed multiple lesions in the left lobe which were not demonstrated by infusion hepatic angiography. In 7 cases (Fig. 4) PTP resulted in falsely negative findings. In 4 cases (Figs. 7, 8) the tumor size was better delineated at hepatic infusion angiography.

Discussion

Postmortem injection studies in livers with malignant lesions (17, 21, 5, 12, 7, 15, 18) have shown that the blood supply of the tumors comes exclusively from the hepatic arteries. The portal vein branches were regularly found to be dislodged or occluded by the mass. Therefore, visualization of the intrahepatic portal vein branches for diagnostic purposes will only demonstrate indirect signs of the expanding and infiltrating tumor. The nature of the lesion cannot be determined, however. Tumor vessels can be revealed only by arteriographic examination. In hypovascular tumors the demonstration of newly formed vessels may be impossible. Liver metastases from primary tumors in the gastro-intestinal tract, such as in the rectum, colon, pancreas, bile duct and stomach, are in the majority of cases of low vascularization and often angiographically avascular in the center (4). The sinusoidal phase of the hepatic infusion study may show these lesions by demonstration of increased accumulation of contrast medium in the periphery, whereas the tumor center cannot be differentiated from the normal liver tissue. In such a case, however, angiography is not definitive and the final diagnosis depends on microscopic verification following fine-needle aspiration biopsy. Whichever access to the portal venous system is used, the diagnostic value of portography depends exclusively on the distinct visualization of the complete intrahepatic portal venous system. Failure to demonstrate the left liver lobe is a common drawback in all direct and indirect methods of portography unless the patient is examined in the prone position. Transumbilical and percutaneous transhepatic portography render possible a selective study of the portal venous system of the left liver lobe. This theoretically may overcome the diagnostic problems in lesions of this lobe due to over-projection of the spine. However, the limited material of this study gives

**Fig. 6B**

PTP — portal venous phase: No space-occupying lesion demonstrated.

**Fig. 6C**

PTP — sinusoidal phase: No space-occupying lesion demonstrated.

evidence that portography is an unreliable method of detecting solitary lesions in the left liver lobe which are smaller than 5 cm. Tumors larger than 7 cm, however, may easily be demonstrated during the portal venous and sinusoidal phases of PTP.

A further drawback of portography in diagnosis of suspected malignant lesions of the liver is its limited value when there are

concomitant vascular changes due to liver cirrhosis. Regional hepatofugal blood flow may result in nonvisualization of large areas of the liver (Fig. 9) (10). The dislocation of portal vein branches due to shrinkage of the liver and formation of regenerative nodules virtually excludes any diagnosis of malignant space-occupying lesions (Fig. 10).

Various authors have demonstrated tumor lesions as small as

Figs 7A–C 63-year-old female with liver cirrhosis, portal hypertension and esophageal varices. Uninodular primary hepatic carcinoma in medial segment of left liver lobe.

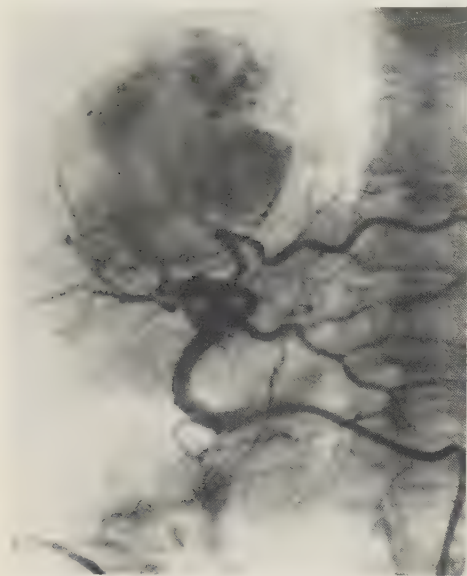


Fig. 7A Infusion hepatic angiography – early phase: Common hepatic artery supplies left liver lobe only. About 7 × 8 cm hypervascular tumor in medial segment of left liver lobe. Abnormal vessels and marked accumulation of contrast medium in tumor area.

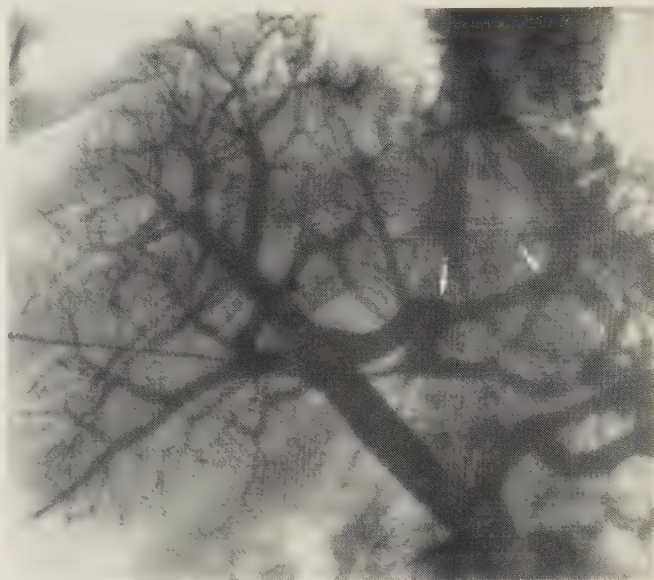


Fig. 7B PTP – portal venous phase: Compression of proximal part of portal vein branch in dorsolateral segment of left liver lobe (→). Estimation of tumor size not possible. Hepatofugal flow of contrast medium to esophageal varices.

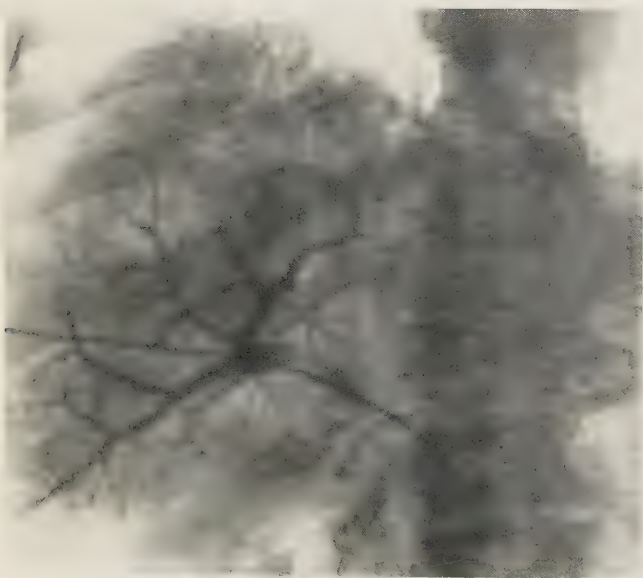


Fig. 7C PTP – sinusoid phase: Irregular and sparse accumulation of contrast medium in both liver lobes precludes detection of space-occupying lesion. Markedly delayed flow in portal vein branches of dorsocaudal segment.

2–3 cm by splenoportography (1, 16) and transumbilical portography (13, 14). According to our experience this is more likely to be the exception than the rule. Based on the findings at hepatic infusion angiography, solitary metastases of approximately 3 cm were not seen at PTP in 4 cases. Multiple metastases of approximately 1–2 cm were seen in most cases, however. The true degree of tumor spread within the liver was better demonstrated at infusion hepatic angiography in all cases but one (Fig. 2). If single 2–4 cm metastases were demonstrated by PTP the diagnostic information was obtained mainly from the sinusoidal phase. Overprojection of portal vein

branches made the identification of space-occupying lesions of this size impossible in the portal venous phase in all cases but one (Fig. 3 b). Tumor lesions larger than 6 cm in the right liver lobe were visualized at PTP as effectively as at infusion hepatic angiography. Tumors as large as 4–5 cm may, however, remain undetected when they are located in the very center of the liver engaging both lobes (Fig. 6).

Arteriography and PTP were performed in 6 patients with primary liver carcinoma. Whereas the size of the tumor(s) in most cases was better delineated at arteriography, PTP excellently demonstrated the main stem of the portal vein and its intrahepatic ramifications (Fig. 11). This detailed morphological information could not be obtained by indirect (= arterial) methods of portography. When liver resection is to be considered in patients with malignant lesions, it is essential to exclude tumor infiltration of the portal vein and its main intrahepatic branches. Compression or infiltration of a major portal vein branch near the liver hilum renders impossible an estimation of the true tumor size. Marked arteriportal shunting from the tumor area may likewise limit the morphological information obtained at PTP.

Conclusion

PTP was non-contributory to the diagnosis in 20 of 21 cases. Infusion hepatic angiography was superior to PTP in the diagnosis of tumor lesions in both liver lobes in 11 of 21 patients. In 9 patients PTP provided approximately the same information as the arteriographic study regarding the extent of the tumor lesions. In one patient PTP was superior to infusion hepatic angiography in detecting multiple lesions of the left liver lobe. The sinusoidal phase at PTP gave superior diagnostic information as compared to the portal venous phase when single small lesions (i. e. below 4 cm in size) were demonstrated. The role of PTP following hepatic arteriography is to provide essential information as to liver tumor resectability rather than to diagnose the presence of tumor.

Figs 8A and B 62-year-old male with liver cirrhosis and multinodular primary hepatic carcinoma engaging both liver lobes.

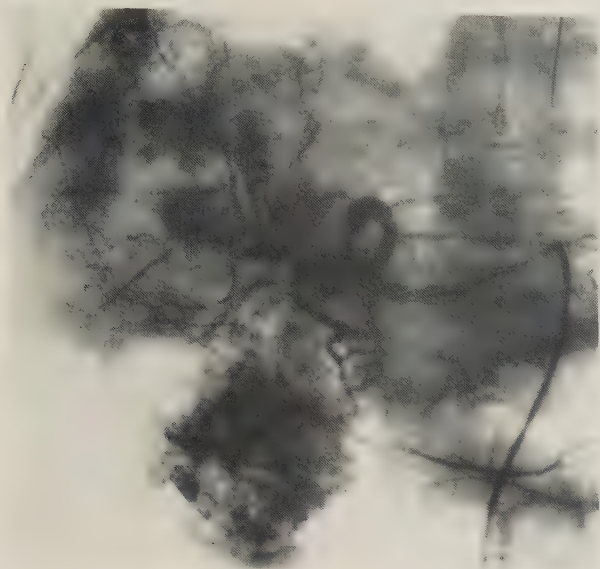


Fig. 8A Infusion hepatic angiography – early phase: Multiple hypervascular, about 2–3 cm tumors in both liver lobes. In addition 5 × 7 cm hypervascular tumor originating from ventrolateral segment of left liver lobe.

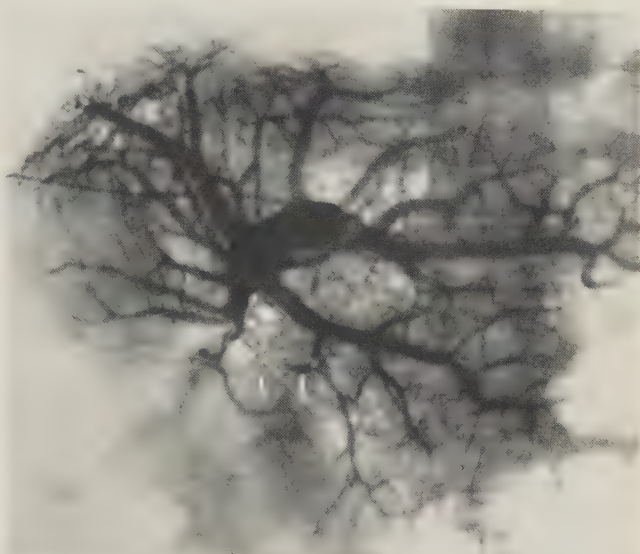


Fig. 8B PTP – selective study of left liver lobe – portal venous phase: Left lobe enlarged with moderate distortion of intrahepatic portal vein branches in medial segment. Tumor in ventrolateral segment dislodges minor portal vein branches (→). Differentiation from vessel displacement due to cirrhosis not possible. No other space-occupying lesions seen.

Figs 9A and B 57-year-old female with liver cirrhosis, portal hypertension and esophageal varices. Liver enlarged and dislodged from lateral abdominal wall because of ascites. No suspicion of liver malignancy.

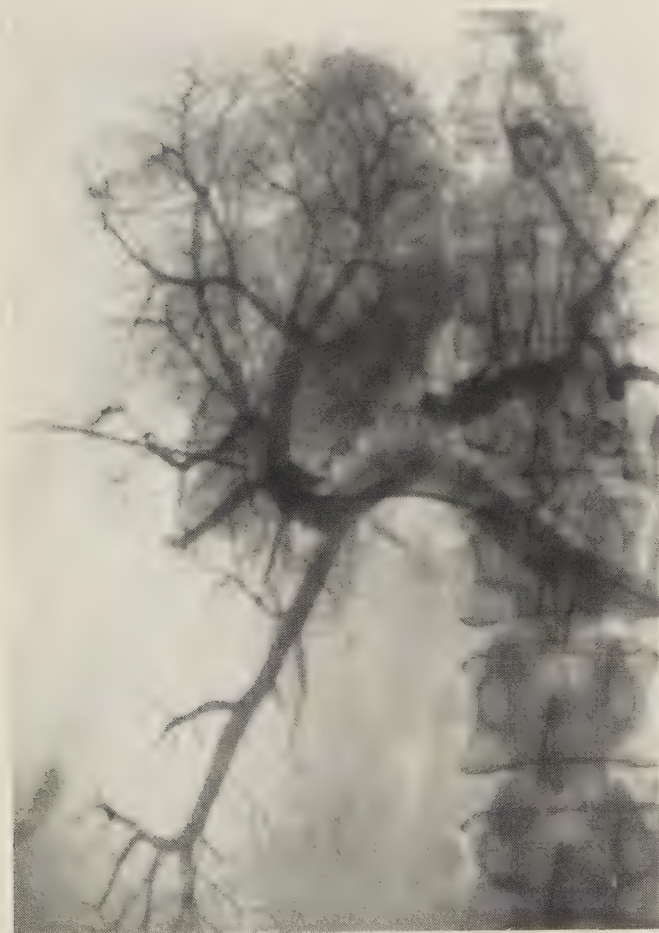


Fig. 9A PTP – portal venous phase: Marked flow disturbance especially in middle and caudal part of right lobe resulting in incomplete delineation of intrahepatic portal vein branches.



Fig. 9B PTP – sinusoidal phase: Irregular and sparse accumulation of contrast medium in sinusoidal phase.

**Fig. 10**

45-year-old male with liver cirrhosis and portal hypertension. No suspicion of liver malignancy. PTP with tip of catheter in dilated splenic vein. Distortion of intrahepatic portal vein branches. Marked enlargement of left liver lobe.

Figs 11 A and B 65-year-old female with uninodular primary hepatic carcinoma in left liver lobe.



Fig. 11A Infusion hepatic angiography – early phase: Huge mass in lateral segments of left liver lobe. No tumor vessels demonstrated.



Fig. 11B PTP – portal venous phase: Catheter tip in proximal part of portal vein. Main stem of portal vein slightly compressed by hepatic mass which contains multiple calcifications. Left main branch of portal vein occluded. Normal ramification in right liver lobe.

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